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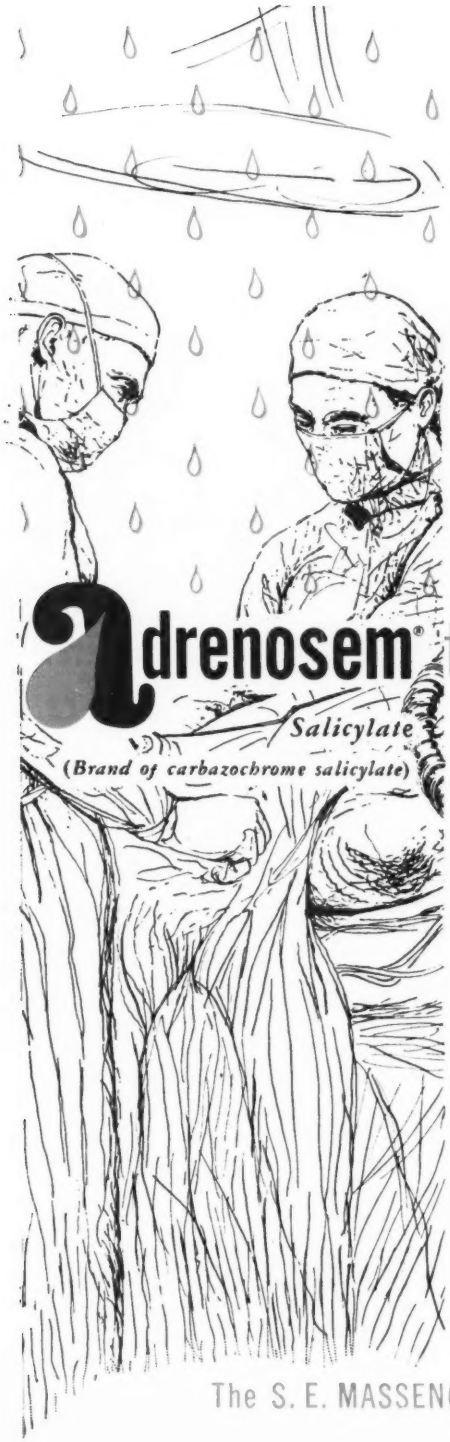

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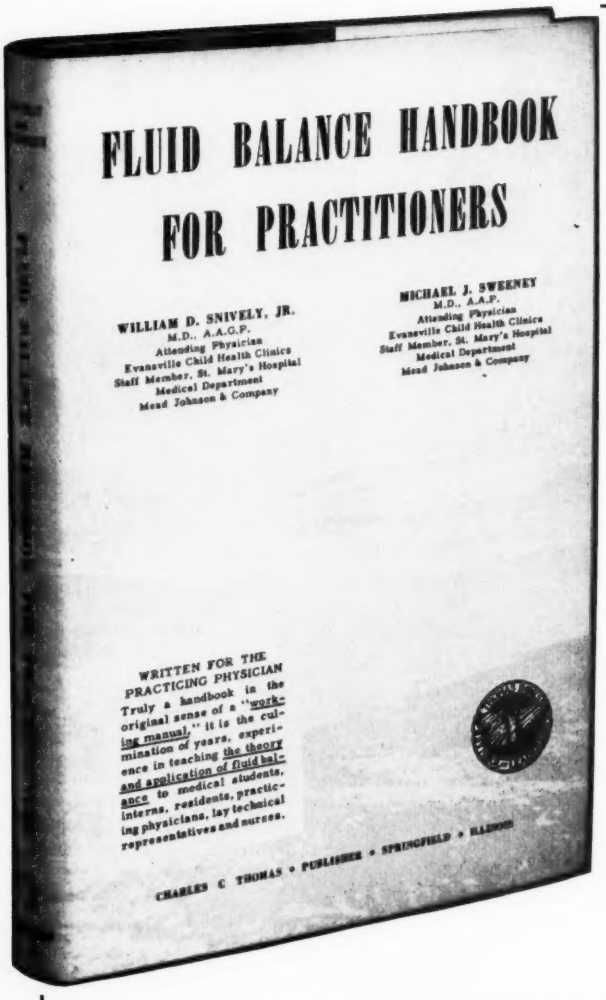
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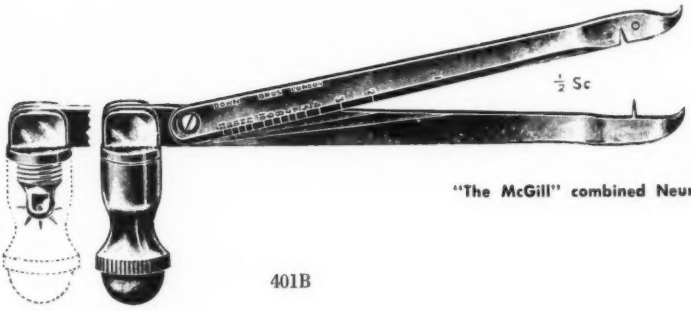

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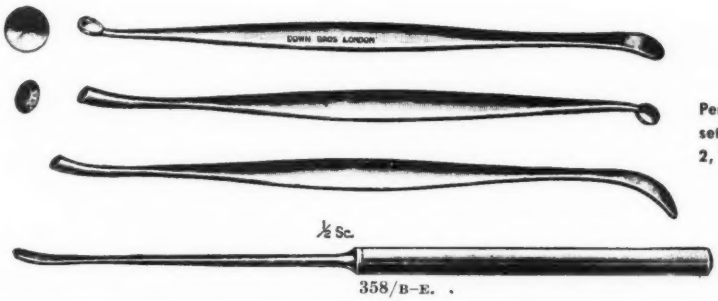

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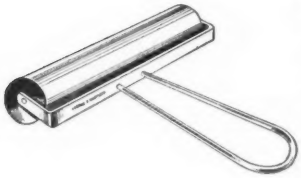

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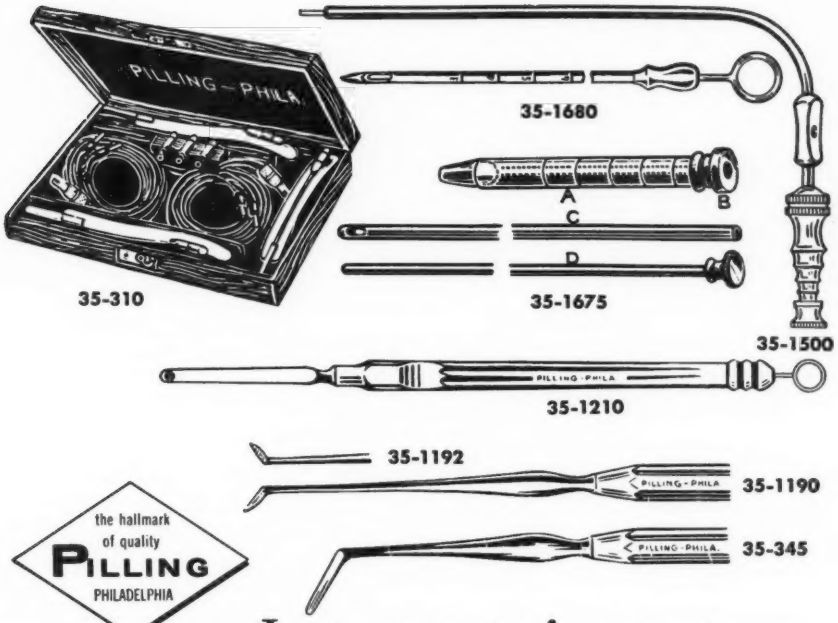

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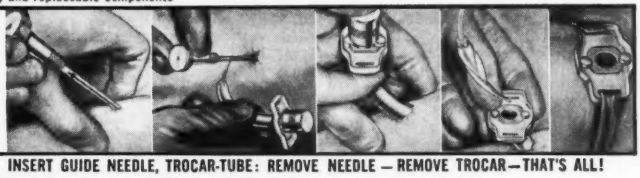

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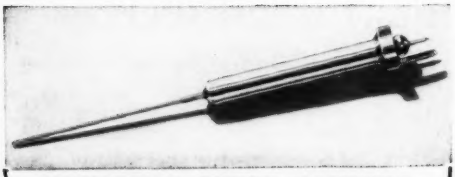

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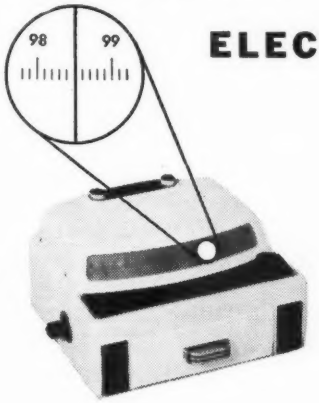

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CEPHALIC BRUITS IN CHILDREN

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(Received for publication April 2, 1956)

THE discovery of an intracranial murmur during the routine physical examination of an apparently healthy infant or young child may startle the examiner and alarm the parents. This is particularly true in recent years because cerebral auscultation has practically ceased to be a part of the clinical physical examination in adults and children, and, therefore, the significance of these bruits may not be appreciated.

Although in the past much has been written on the subject of intracranial bruits in infants and young children, this interesting material actually has been collected in only a few places, and it seemed of value to record a review of the writings of earlier physicians concerning the significance of intracranial bruits in infants and young children.

Writing in the *American Journal of Medical Sciences* in 1838, Fisher[8,9] stated that 5 years previously he had read a paper on the cephalic bellows sound and considered that this physical finding might play an important role in the diagnosis of cerebral diseases as well as thoracic diseases. He described cephalic sounds associated with the heart, with respiration, with the voice and with deglutition as they are developed in normal children at rest or asleep and prior to closure of the anterior cranial fontanelle.

The cephalic bellows sound (cranial bruit), Fisher observed, was related to "simple congestion of the cerebral organs," chronic hydrocephalus, acute inflammation of the brain, suppuration (brain abscess) and induration of the brain accompanied by effusion into the ventricular system and at the base and also by immediate pressure on the brain.

In 1839 Smyth[34] reported a case of chronic hydrocephalus relieved by tapping and stated that in a former paper, written in 1837,[33] he had alluded to the cranial systolic murmur of chronic hydrocephalus.

The observations of Fisher[8,9] were quickly picked up by physicians in Europe and many reports and ideas were contributed mainly by French and German physicians.

Versini,[39] in 1839, translated Fisher's observations into French, even to citing Fisher's conjecture that the "bruit de souffle" had its origin in the arterial trunks at the base of the skull where the caliber of the intracranial arteries is compromised by the skull itself.

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* The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

In 1841 a treatise on auscultation was written by Barth and Roger[1] who although they accurately summarized Fisher's work, even to his conclusion that the cephalic bruit did not exist in a normal state and was pathologic in import, commented that to that time no trained observer in France had been able to confirm his results.

Whitney[40] reported that he had encountered this cephalic systolic bruit in association with eight conditions. He listed an aneurysm of the basilar artery, and mentioned a statement by Tweedie[38] to the effect that probably many times use of a stethoscope in cases of aneurysm would reveal a bruit (bellows murmur) and would facilitate diagnosis. This might have been the beginning of the misconception, so aptly denied by Beadles[2] at a later date, that intracranial aneurysms frequently are accompanied by a bruit.

The early dispute over the significance of intracranial noises was not without its humorous side, for Kühlbrand,[17] in 1846, reported the case of a woman who was using her lower jaw and tongue to cause noises which were heard in the head and which had caused much concern among the local physicians and had deceived many of them.

One of the earliest definitive articles from Germany, perhaps the first and, according to some, the most complete, was contributed by Wirthgen[41] in 1855. He suggested that there was an anatomic-physiologic reason for the intracranial bruit in some children and that the bruit was the shock of the carotid pulse transmitted along the great vessels of the skull. The skull bones increased the resonance and this was the cause of the intracranial bruit. Wirthgen stated that the reason a bruit may not be heard in very, very young children is that the bones are too loosely united to provide resonance.

Hennig,[12] writing a year later, was of the opinion that the source of the cephalic blowing sound was in the arterial system, but he did not think the sound was the result of vibration of the arterial walls themselves, or of the noise they communicate to the cephalic mass or to the walls enclosing the cephalic mass. He concluded that if the arterial system is the source of the noise, it must be the intracranial venous system that transmits it.

In 1859, in a rather extensive article, Rilliet[25] summed up much of the material on cranial murmurs and added some thoughts of his own. To that time, it was apparent that there was a complete divergence of opinion between American and German physicians on two points. Wirthgen[41] and Hennig[12] were certain that the cephalic blowing sound (bruit de souffle) in infants could be observed in healthy children, whereas Fisher,[8] the American, was equally certain that the noise did not exist intracranially in a healthy young individual. The German physicians Wirthgen and Hennig claimed that the cephalic bruit disappeared as the intracranial pressure increased. Fisher,[8,9] on the other hand, believed exactly the opposite: that the cephalic blowing sound in children was caused by increased cerebral pressure.

Then Rilliet[25] added his opinion to the controversy and stated that increasing intracranial pressure caused the cephalic bruit to cease. He stated also that chronic pressure could cause the bruit to disappear and disagreed

with Hennig and Wirthgen that the cerebral tension needed to increase rapidly to have this effect.

Roger,[29,30] writing at about the same time as Rilliet, that is, 1859–1860, analyzed some 300 observations on head noises and came to the following conclusions: that on careful auscultation of the head only a single noise, the cephalic blowing sound, would be detected. He did not feel that there was cerebral egophony, which might have been characteristic of either cerebral effusion or apoplexy, or any other intrinsic noise in the head. Furthermore, he expressed the opinion that no affection of the head could be accurately diagnosed on the basis of a bruit de souffle. He stated that the bruit de souffle is a noise frequently associated with chloro-anemia in very young patients, but rarely with cerebral disease. He also noted the common occurrence of this cephalic bruit in children having rickets, and emphasized the fact that rickets should not be considered as a disease of the skeletal system but of the blood; thus he conveyed his idea of the relation between cephalic bruit and anemia in infants.

In 1861, Henoch[13] stated that the important decision to make was whether or not a systolic blowing murmur in the head of infants was of pathologic import. After a review of the available material Henoch decided that the cephalic bruit de souffle in infants was pathologic and did not represent a normal physiologic mechanism.

Somewhat later, in 1877, Jurasz[16] concluded that the systolic murmur in the brain occurs in children from the third or fourth month of life until the fourth to sixth year. He considered that the bruit was in direct relation to the development of the base of the skull, especially that of the carotid canal and the foramen spinosum. Jurasz stated that these murmurs are physiologic in origin and only rarely have pathologic significance. For this reason, he did not consider systolic brain murmurs in children as diagnostic criteria in cranial disease.

One year later Epstein[6] presented the view that brain noises in children were an expression of the carotid artery, and also of the noises arising from the large intracranial arteries. The most favorable age for the origin of the bruit was the last half of the first year of life and the entire second year. In his experience Epstein felt that the exceptional boundaries might include the third month to the sixth year of life. He stated that childhood, particularly the first year of life, offered an especially favorable chance for the origin of these vascular noises in the organization of the child's beginning. He concluded that brain noises were not a pathologic happening.

In 1880 Osler[22] wrote his classic article "On the Systolic Brain Murmur of Children," which most subsequent reviewers on this subject have used as their basic reference. Osler wrote his article in an effort to redirect medical attention to auscultation of the head and its importance. He presented the varying opinions of many of the afore-mentioned authors, but did not venture a statement as to the explanation of the systolic murmur encountered in some young children.

A year later Tripier[37] stated that one must, by exclusion, place the origin

of the systolic blowing intracranial murmur in the terminal portion of the internal carotid artery at the level of the point of entrance into the cranial cavity. In anemia resulting from hemorrhage, cachexia and chlorosis, the cephalic bruit is encountered while the symptoms of anemia are particularly intense and of long duration. He stated that a cephalic bruit without a murmur at the base of the heart and especially without anemia should cause the examining physician to consider the possibility of compression of the internal carotid artery at the level of its terminal portion.

It is important to distinguish between the cephalic blowing sound in which the bruit is continually being reinforced, Tripier stated, and that in which the bruit appears intermittently, produced either by communication of the carotid artery and cavernous sinus or by aneurysms of the carotid or ophthalmic arteries, because in all these cases he felt characteristic signs would be present in the orbit on the same side.

Somewhat later Rohde[31] quoted much of the available controversial information, and listed Roche's[28] eight points published in 1859 which seemed of interest.

Roche had stated that brain noises are very common in small children when they have anemia. During dentition and the first year of life anemia is common, and so, according to Roche, are brain noises. They are prominent also in whooping cough, but they are not common in rickets. As previously mentioned, Henoch considered brain murmurs to be symptoms of rickets.

A little earlier (1863) von Rittershin[26] had explained that brain murmurs are found only in children with open fontanelles and therefore were not of any diagnostic significance. In his review Rohde finally concluded that fontanelle noises were pathologic occurrences.

It is of interest, however, to note that in his *Lectures on Children's Diseases* Henoch[14] stated that he and Wirthgen directed attention to the physiologic occurrence of this bruit between the twenty-second or twenty-third month of life at the time of closure of the fontanelles by ossification. Henoch, in agreement with Roger,[29,30] stated the opinion that the so-called brain murmur was of no interest from the clinical point of view.

Until Beadles'[2] article in 1907 on aneurysms of the larger cerebral arteries, contributions by Richardson,[24] Teleky,[36] Robertson,[27] Lane,[18] Oliver[21] and others were of some interest but added little to the over-all knowledge of the subject of brain murmurs in children.

Beadles' paper was of particular interest because he reviewed 555 cases of intracranial aneurysm. According to Parker[23] he made the following statement: "I doubt if more than a couple of living physicians have ever seen a case (of aneurysm) in which a murmur has been audible, and in which the evidence is conclusive that the patient had a true aneurysm of a cerebral artery."

Some years later (1921) Still[35] wrote on "Cephalic Bruits in Children." He stated that most textbooks passed over the subject of intracranial bruits in children, partly because the bruits had no recognized pathologic significance.

Still reviewed much of the literature referred to in this report, but stated that his chief interest was centered on the bruit heard in children whose fontanelles were already closed. He mentioned that he was entirely unable to connect the cephalic bruit with any particular morbid condition among the 30 children in whom he detected it. In the 30 cases many diseases were represented: scurvy, epilepsy, achondrodysplasia, nephritis, asthma, congenital syphilis, congenital heart disease, acidosis and dyspepsia; four children did not have any disease at all.

Cushing and Bailey[4] mentioned that they considered auscultation of the head, used to detect bruits in children, to be a lost art.

Hamburger's[11] excellent paper on intracranial bruit was not published until 1931. He stated that cephalic murmurs in children prevail particularly during the second year of life, and during this period they occur about two and a half times as frequently as in the first year of life. Although anemia and rickets were pointed out as causative agents of head murmurs of infants, anemia, whether it was accompanied by rickets or not, was not a necessary part of the picture. These infantile bruits can be heard in entirely healthy children, and, according to Hamburger, there is no record that a murmur may have persisted until adult life.

Other types of murmurs that may be encountered are those of arteriovenous fistulous origin and those associated with rare cases of carotid aneurysm, occasional orbital neoplasms and still rarer cases of true aneurysm of the ophthalmic artery. Another variety of cephalic murmur of intracranial vascular origin discussed by Cushing and Bailey[4] is that which accompanies cerebral aneurysmal angioma.

In addition to these afore-mentioned bruits, murmurs have been heard in the presence of intracranial lesions, tumors, cysts and so forth.[15,20,32]

Sears,[32] in an extensive article published in 1938, considered the mechanism of the production of the cephalic bruit in childhood as unknown, but reiterated the following suggestions: that the condition is attributable to a temporary stenosis of the internal carotid artery in the carotid canal because of the increase in the volume of the rest of the artery that is out of proportion to the rate of growth of the bony canal, or a similar state of affairs causing an increased tortuosity of the artery at the base of the skull in childhood that tends to straighten out as the skull grows and thus abolishes kinking of the artery.

According to Sears, evidence was not available to show that the bruit persisted until adult life. He concluded that the appearance of a cranial bruit in childhood in the absence of any other neurologic lesion is of no pathologic significance, but agreed with Still that it is useful to know of its occurrence and its innocence.

Dalsgaard-Nielsen[5] based his report on the cases of 2,000 patients on whom systematic auscultation had been carried out. He stated that a vascular sound is heard in a good many normal children, and the maximal incidence is encountered in the age group from 6 to 16 months. After this the incidence rapidly drops and no vascular sound is heard in older children

or adults. He stated that the frequency of vascular sound is slight in children less than 6 months of age, and in the author's series vascular sound was not heard in infants less than a month old.

Dalsgaard-Nielsen described the vascular sound as being heard on auscultation over the anterior fontanelle in children and the disappearance of the sound at the age when the infant's fontanelle closes. He described the sound as faint, soft and blurred without color or distinct limits. The sound also apparently is not permanent and not infrequently, according to the author, subsides during auscultation. He related that this cranial bruit is a physiologic vascular sound that is heard in healthy young children and does not arise in the arterial part of the cranial circulatory system but is derived essentially from the venous portion of the cranial vessels. On the other hand, he recognized that an intracranial vascular sound in older children or adults is definitely of pathologic significance.

Interestingly enough, Fay,[7] in 1940, briefly discussed the diagnosis of intracranial disease by the use of auscultatory percussion of the skull, and then in 1941 Marie and co-workers[20] reported the discovery of a tumor by auscultation in a child 2 years and 9 months of age.

A review of the literature on cephalic bruits was presented by Mackby[19] in 1942. Ford,[10] in 1944, discussed the subject of cranial bruits in childhood briefly, but drew largely on reports by Osler and Hamburger.

Ford stated that a number of different types of intracranial bruits may be encountered during childhood. Some of these are indicative of serious conditions and others are apparently without pathologic significance. He discussed the systolic murmurs of early childhood, stating that in his opinion anemia is the usual cause of these benign murmurs. He made an interesting point, however, in stating his hesitancy to give a good prognosis to the parents if the infant should have an intracranial bruit and normal hemoglobin.

The most recent contribution to the subject of auscultation and intracranial disease at the time of this writing is that of Burklund,[3] published in June, 1953; this article reiterates many of the points already touched on in the articles mentioned herein.

CONCLUSIONS

On the basis of many detailed reports, several conclusions seem valid concerning the cranial systolic bruit encountered in infancy and early childhood. Through the years opinions have varied greatly as to whether these bruits are physiologic or pathologic, arterial or venous. The actual incidence among children is unknown, although conjecture has established the occurrence of this entity as both frequent and rare.

Certainly very little basic physiologic research has been reported to elucidate the mechanisms and significance of this phenomenon. Almost universal agreement exists, however, as to the age of occurrence and maximal incidence and length of duration. In addition there is wide accord as to the rarity of persistence of this cranial symptom until adult life.

In the opinion of the reviewer, it seems likely that if auscultation is carried out in infants and younger children with the idea of detecting such cranial systolic bruits these sounds might be encountered more frequently. In the neurologically normal infant showing adequate progressive development the examiner might assume the benignity of the symptom, but should reserve his final opinion until time and growth have proved his assumption to be correct. Auscultation of the carotid arteries in the neck should always be done because sometimes the bruit may be heard here and transmitted to the head. Anemia as a contributing factor might well be remembered, but the presence or absence of this laboratory finding should not encourage or dishearten the physician in his over-all appraisal until continued growth of the child has elucidated the problem. Ford's[10] warning concerning the presence of a cranial bruit and normal hemoglobin seems worthy of reiteration.

The discovery of an intracranial systolic bruit in a neurologically normal infant or young child should be deemed worthy of notation and judicious observation, with the mental reservation on the part of the physician of a usually favorable outlook. Once again the words of Osler, Cushing and many others, that the art of cranial auscultation has been largely overlooked, bear repetition.

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THE DIAGNOSIS, TREATMENT, AND PROGNOSIS OF TUMORS AFFECTING THE SPINAL CORD IN CHILDREN

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TUMORS of the spinal cord in children are relatively infrequent. During the past 25 years at the University Hospital and the Graduate Hospital of the University of Pennsylvania, a series of 409 spinal cord tumors were treated, and of these 30 were in children under the age of 15 years. The ages at which these tumors occurred are shown in Table 1. It should be pointed out that the present study deals with only primary tumors involving the spinal cord, with the exception of 2 epidural abscesses. Thus, metastatic tumors or seeded tumors, such as those of cerebellar medulloblastoma, have not been included. As far as the site of spinal cord tumors in children is concerned, it has been noted in our series that their frequency at different locations corresponds to the relative length of the spinal segments (Table 2). It is curious that in children one of the major difficulties in diagnosing these lesions is the fact that symptomatology is usually first

TABLE 1

Incidence of 409 spinal cord tumors at various ages

Years	No. of Tumors
0–9	10
10–15	20
16–19	26
20–29	54
30–39	54
40–49	84
50–59	100
60–69	53
70–79	7

TABLE 2

Location of 30 spinal cord tumors in children

Cervical	8
Thoracic	14
Lumbar	5
Sacral	3

noted following a relatively minor injury. Hence, it is common that the actual onset is clouded and unnoticed and the early symptoms are associated with the usually totally irrelevant trauma. We found that symptoms were rarely present for more than 6 months, but again it was difficult to be sure because of the rapid growth and development of the child and the ability of the child's nervous system to compensate for mild neural damage. In 15 of our cases the clinical symptoms were present for 4 to 6 months; in 11 patients there was a 2 to 3 months' duration and in 4 patients the symptoms were of less than 1 month's duration.

DIAGNOSIS

Once the suspicion has been aroused that possibly the child is presenting symptoms of spinal cord tumor, diagnostic measures should be undertaken without delay. Of primary importance is the neurologic examination which should be carried out preferably by a neurosurgeon or neurologist. This should include a careful search for the presence of muscular weakness with specific localization of any weakness to the groups and single muscles involved. There should be a meticulous sensory examination, including tests of the senses of pain, temperature, and light touch, which is best done with von Frey hairs when the child is able to cooperate. We emphasize the importance of the sensation of light touch because we have found frequently that a level to pain and temperature may be as much as five segments lower than the level to light touch. Tendon and skin reflexes should be tested and discrepancies in laterality noted, and, likewise, an attempt should be made to elicit the anal and cremasteric reflexes, the absence of which is often associated with lesions of the cauda equina. It is important to determine whether or not there is involvement of the bowel and bladder. This may be difficult in a child but is essential to future evaluation. The neurologic examination should usually determine the presence of a tumor, the approximate level of the lesion, and whether or not there is evidence of spinal shock or spasticity. For the sake of simplicity we feel that spasticity is best defined as an increased resistance to passive movement in the limb examined, plus hyperactive tendon reflexes, usually associated with a positive Babinski sign and ankle clonus.

Much added information may be obtained at this stage by electromyography and strength-duration curves. These are not always necessary but frequently aid in detecting specific motor weaknesses by the absence of normal motor unit firing in the electromyogram, and the determination of whether or not atrophy is caused by disuse or a lower motor neuron lesion in the case of chronaxies. When there is known dysfunction of the bladder a preoperative cystometrogram is essential in order to evaluate the full extent of the damage, and to serve as a base line for possible postoperative improvement. Whenever there are signs of atonic bladder, we feel that it is best to place an indwelling Foley catheter in the bladder as soon as the diagnosis is made. There is adequate evidence for both the use of tidal drainage,

or the use of intermittent drainage by keeping the catheter clamped and releasing it every 4 hours. The fact that tidal drainage possesses certain similarities to the normal physiology of bladder distention makes it somewhat the more favorable procedure. If there is a severe degree of paralysis present, one should consider the possibility of breakdown of the skin and the advisability of using an oscillating bed with a variable pressure mattress to avoid venous congestion or excessive pressure at any one point.

In our series of cases, the commonest preoperative neurologic finding was a sensory level to pin-prick, which was present in 18 patients (Table 3). The initial symptom of greatest frequency was that of pain, which was present in 15 patients, including 3 with intramedullary tumors. The fact that an

TABLE 3

Frequency of preoperative neurologic findings in 30 cases of spinal cord tumor in children

Preoperative Findings	Location of Tumor											
	Extradural				Extramedullary				Intramedullary			
	C	T	L	S	C	T	L	S	C	T	L	S
Spinal shock	1	2			1					2		
Spasticity of legs*	1	1			1	6			2	1		
Bladder or bowel dysfunction	2	4	2	1		2	3			1		
Pain as initial symptom		2	2	1	1	2	3	1	2	1		
Weakness as initial symptom	1	3			1	4			2	1		
Sensory change as initial symptom					1					1		
Paraplegia (total)	3	4	1			1	1		1	2		
Sensory level to pinprick	2	4	2		1	4	1		2	2		

* Bilateral Babinski, increased tendon reflexes, ankle clonus, and increased resistance to passive movement.

intramedullary lesion may produce pain is brought out by our series of syringomyelia in adults, which showed that in more than half the cases pain was a common early symptom.

For the purpose of elucidation we are presenting 2 typical cases to illustrate some of the points mentioned.

REPRESENTATIVE CASES

Case 1. R.O., a 12-year-old white male, was admitted to the Hospital of the University of Pennsylvania on June 11, 1952 with a chief complaint of paralysis of the legs of sudden onset. He had been in his normal state of health until 1 month prior to admission when he first noticed a slight numbness of the right foot, but he was able to continue his daily activities without difficulty. One week before admission the patient fell from his bicycle when he went over a stone wall. There was no immediate injury apparent but, within 24 hours, he noted numbness of the left foot. The same day his mother first noticed that he tended to drag his legs a bit, and that night it was discovered that he was unable to void. He was admitted to a local hospital and transferred to the University Hospital 3 days later.

Examination. On admission the patient had a flaccid paraplegia. There was a sensory level up to T9 bilaterally with loss of all modalities. There was loss of all reflexes of the lower legs and the lower abdominal reflexes were absent. The cremasteric reflexes were diminished but the anal reflex was normal. The patient showed priapism and some nuchal rigidity.

Roentgenograms of the spine showed no abnormalities. Lumbar puncture on the day of admission showed an initial pressure of 140 mm. of water, and revealed clear cerebrospinal fluid. Queckenstedt's test was negative and did not suggest a block. The following day myelography was done and no block was noted by fluoroscopy. The myelograms showed a fusiform dilatation of the opaque column extending upward at the level of the 9th thoracic interspace. Because the films were interpreted as indefinite, myelography was repeated 12 days later when an additional 3 cc. of dye were used. At this time 9 cc. were injected, and again the changes suggested a swelling or tumor at the level of T9 or T10.

Operation. An intramedullary spinal cord tumor was found. It extended from the 8th to the 10th thoracic segment of the spine. About 80 per cent of the tumor was removed by splitting the posterior columns and sucking out the intramedullary tissue.

Pathologic Diagnosis. Ependymoma.

Postoperative Course. The patient regained some sensation but no motor function of the legs. He received intensive roentgen-ray therapy consisting of a tumor dose of 3600 r. He survived 18 months after operation and died in his home of extensive tumor recurrence.

Case 2. G.S., a 15-year-old school boy, was admitted to the Hospital of the University of Pennsylvania on April 9, 1941, with a complaint of numbness and weakness in both legs. He had been in his normal state of health until 1½ years previously when he had three or four attacks of dizziness associated with epigastric pain and pain in the lower ribs. Six months prior to admission he began to complain of attacks of pain over both the anterior and posterior portion of the lower ribs. These attacks of pain increased until they came on several times weekly. Six weeks before admission the patient began to note numbness and weakness in his legs, although on closer questioning it was found that the legs had been getting slightly weak for a period of 1 year. One month prior to admission the patient went to bed since his legs had become so weak that he could hardly walk. Two weeks later he was admitted to his local hospital where roentgenograms of the spine and lumbar tap were reported to be normal. From there he was transferred to the University Hospital.

Examination. On admission the patient had a severe paraparesis. There was a sensory level to T8 for all modalities including position sense. The lower abdominal reflexes were absent and there was a bilateral Babinski reflex with ankle clonus. The muscle tone in the legs was increased, with hyperactive tendon reflexes. There was poor anal sphincter tone and there was some urinary retention, necessitating a catheter. Lumbar puncture revealed xanthochromic fluid with a pressure of 170; protein was 575 mg. per cent.

Queckenstedt's test indicated a complete block. Roentgenograms of the spine showed some changes in the bone at T10, suggesting a degenerative disease. A myelogram was done using Lipiodol, and this revealed a complete block at the level of the spinous process of T7.

Operation. On April 10, 1941, laminectomy of T5, 6, 7, and 8 was done which re-

vealed a large epidural tumor involving the bone at T6-T7. The majority of the tumor was removed, including the involved bone, but it was impossible to extirpate it completely. The dura mater was opened; there was no intradural extension of the tumor. It was then sutured without further manipulation.

Pathologic Diagnosis. Giant cell tumor of bone.

Postoperative Course. The patient did well and had very little difficulty in voiding. At the time of discharge on April 27, 1941, there was still a partial sensory level to T9 bilaterally. Some movement of the legs had returned. When last heard from, in November, 1954, the patient's condition was completely normal. He was attending Theological Seminary and leading a full time life as a student, engaging in extracurricular sports. He had no return of any of his previous pain or weakness

Comment. Case 1 illustrates the insidious onset that may occur in children and the ease with which these tumors may be associated or confused

TABLE 4

Diagnosis of spinal cord tumors in children

18	Myelography required
11	Routine roentgenograms diagnostic
17	Queckenstedt positive
7	Queckenstedt negative
6	Queckenstedt not recorded
13	Protein >100 mg.%; 11 had positive Queckenstedt

with trauma. In this particular case it is also apparent that spinal shock may result when a tumor is growing rapidly, possibly because of sudden impairment of the vascular supply of the cord with resulting ischemia. In Case 2 it is noteworthy that with epidural tumors pain can be the initial symptom because of dorsal root compression. As these tumors grow, however, they tend to compress the cord, producing weakness and numbness of the extremities. Case 2 also illustrates the possibility of a long-term cure in cases of benign tumor, in spite of an incomplete removal.

In our group of 30 tumors, 11 could be diagnosed by routine roentgenograms of the spine (Table 4). Of the 11 tumors that produced radiologically visible changes in the bone, 6 were extradural, 3 extramedullary and intradural, and 2 were medullary. The changes commonly noted were gross destruction of bone, widening of the interpedicular space, or erosion of the pedicles. Fig. 1 shows the marked bony destruction of the sacrum in a case of giant cell tumor.

LUMBAR PUNCTURE

Although generally considered a relatively mild diagnostic study, we have become more and more aware that puncturing the subarachnoid space in itself may lead to severe neurologic deficit in the presence of a high spinal cord tumor or cervical disc. Thus, in a small percentage of cases, we have noted rapid onset of hemiplegia or quadriplegia within a period of 2 to 8 hours following lumbar puncture. It is for this reason that we believe lum-

FIG. 1. Erosion of the left side of the sacrum by a giant cell tumor.

bar puncture should be done cautiously, when one is suspicious of spinal cord tumor, and that this procedure should be done after plain roentgenograms of the spine have been taken. When manometric studies or xanthochromic cerebrospinal fluid indicate the presence of a block, a small amount of Pantopaque injected at this time will eliminate the need for an additional lumbar puncture. In this way it is often possible to combine myelography with the initial lumbar puncture. One must keep in mind, when there is a deficient circulation of spinal fluid as with a complete block, that the irritating qualities of any contrast medium will be increased because of the more or less focal position of the dye. At present, Pantopaque is the contrast medium of choice.

The Queckenstedt test was positive in 17 of the 24 cases in which it was recorded. The protein content of the cerebrospinal fluid was elevated in 13

cases (Table 4). The value of myelography in confirming the neurologic findings cannot be overestimated. Meticulous as one is with the neurological examination, it is common to be one or two segments off in outlining the lower pole of the tumor. With myelography this is done with great precision and permits a minimal-size incision for maximum exposure of the tumor. Fig. 2 shows the defect in a case of an extradural tumor at T4. An intramedullary cord tumor outlined by Pantopaque at T10 is shown in Fig. 3. In some cases of spinal cord tumor it may be desirable to outline the upper limits of the lesion and in these cases we do a cisternal puncture and put in 2 or 3 cc. of radiopaque dye from above the tumor. The disadvantage of this diagnostic procedure is that there is a certain danger associated with cisternal puncture and it is almost impossible to remove the contrast medium before the operation. In Fig. 4 is shown a case in which cisternal myelography has outlined the upper level of a tumor at T1.

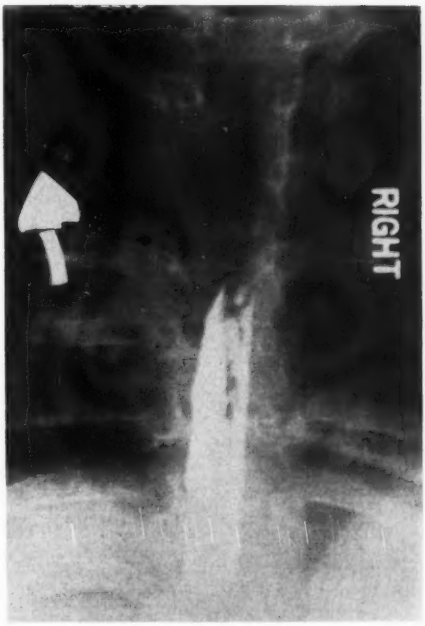

FIG. 2. Myelographic block at T4 caused by an extradural tumor.

PATHOLOGY

The pathologic types of the 30 spinal cord tumors, with reference to their location, are shown in Table 5. For the purposes of this study the various types of gliomas, neuroblastomas, lymphoblastomas, and sarcomas have been classified as malignant. Although it can be seen that all the intramedullary tumors were malignant, little in the way of further conclusion can be drawn concerning the relevance of tumor cell type and the position of the lesion. One may say that it is more common for a tumor to be benign if it is in an extradural or extramedullary position. It is also interesting that some tumors, such as ependymomas, may occur either as intramedullary tumors, or as extramedullary lesions in the cauda equina. Similarly, the neurofibromas were found to occur either as an extradural lesion, or as an intradural but extramedullary type of lesion. In this series, 17 tumors were benign and 13 were found to be malignant.

It is interesting to note that meningiomas were our commonest type of tumor as well as being the commonest intradural tumors. This has not been

FIG. 3. Myelographic defect caused by an intramedullary tumor at T10.

true in other series, however, for Svien, Thelen and Keith[2] found no meningiomas in their series while Ingraham and Matson[1] found only 2 meningiomas out of 63 tumors. Similarly, Ingraham and Matson[1] found that teratomas and dermoid cysts were their commonest pathologic types, comprising 33 per cent of their entire series while we have none of these types. This illustrates the great variation from clinic to clinic, and shows the difficulty of comparative studies on a statistical basis.

SURGICAL TREATMENT

When the tumors are extradural it is our practice not to open the dura mater, for if this is done some of the blood vessels in the dura mater and arachnoid will be automatically sacrificed, and, when there is already neurologic deficit, it is best to conserve every bit of both the arterial and venous supply. Similarly, when the tumors are intramedullary, as a rule we do not open the arachnoid over an area wider than is necessary. First of all, the arachnoid serves as a protective covering over the cord and normal spinal fluid pathways are more readily re-established when it is left intact; and secondly, a goodly number of fine blood vessels run in the arachnoid which frequently must be sacrificed during the course of its opening. A third advantage to be gained from leaving the arachnoid as intact as possible is that, when it is

FIG. 4. Cisternal myelogram showing block at upper pole of tumor at T1.

found advisable to leave the dura mater open, one then has the secondary protection of the arachnoid over the cord. We never hesitate to leave the dura mater open if the cord is swollen when we have been dealing with an intramedullary tumor that is likely to recur. The added decompression is

TABLE 5

Pathologic diagnoses in relation to position of spinal cord tumors in children

Extradural—12		Intradural (Extramedullary)—12		Intramedullary—6	
Giant cell tumor	3	Meningioma	5	Astrocytoma	3
Sarcoma	2	Neurofibroma	2	Ependymoma	2
Abscess	2	Neuroblastoma	1	Spongioblastoma	1
Neurofibroma	1	Ependymoma	1		
Neuroblastoma	1	Cystic tumor	1		
Tuberculoma	1	Vascular malformation	1		
Cyst	1	Neuroepithelioma	1		
Lymphoblastoma	1				

well worth while and we have not found any harm to come from this procedure. The greater the neurologic damage before operation, the more carefully should one avoid trauma in approaching these tumors, for part of the venous drainage of the cord occurs through venous sinuses which involve the bone, so that even increased removal of bone may cause deficiency in the venous drainage of the cord. Much bleeding is, of course, unlikely to occur with these lesions but, on the other hand, considerable venous bleeding may be encountered by going too far laterally and becoming involved in the epidural veins. Occasionally this is unavoidable and here it is wise to suture the dura mater back tightly over a thin strip of gelfoam placed in the lateral gutter.

As mentioned previously, it is often difficult to diagnose tumors of the spinal cord in children because of the constantly changing and developing nervous system of these little patients. For this same reason, it is also easier for them to recover from a paraparesis than an adult. It is difficult to use strongly positive statements in the matter of prognosis, but certainly if a tumor is extradural or extramedullary, the prognosis is much better for these children. Although they seem to recover from paraparesis usually in a satisfactory manner, when there is a complete paraplegia the outcome is extremely grave. Thirteen of our patients were completely paraplegic at the time of surgery. Of these 9 had a flaccid type, and 4 had a spastic type of paraplegia. The only one of these children who improved postoperatively was one who showed flaccid paraplegia. The spastic paraplegias were accounted for by tumors in the upper and mid-thoracic area, whereas the flaccid paraplegias were caused by tumors distributed throughout various spinal levels. It is for this reason that we believe these spinal cord tumors in children should be regarded as an acute surgical emergency, as one would a brain tumor with increased intracranial pressure.

FINAL RESULTS

In Table 6 are outlined the ultimate results in our series of 30 patients. There were 2 operative fatalities (6.7 per cent). One of these patients had an intramedullary tumor in the high cervical region, and the other had an epidural spinal abscess of extreme degree which involved the entire cord as high as the cervical region. Both patients died of respiratory failure. It may be said that some of the patients were operated upon before the days of antibiotics.

TABLE 6

Final results in 30 cases of spinal cord tumor in children

Malignant tumors	40.0%
Benign tumors	60.0%
Operative mortality (30 days)	6.7%
Case mortality	37.0%
Working or asymptomatic at end of 5 years	30.0%

In a 5-year follow-up study it was found that 9 (30 per cent) patients were totally asymptomatic, and either working or attending school full time. Nine patients were followed for more than 10 years, and of these 7 were asymptomatic and either in school or working on a full-time basis. The 9 patients who were followed for 10 years or more, and who were placed in the class of 10-year cures, all had benign tumors except for 1 who had an ependymoma of the cauda equina which had been partially removed, with the dura mater left open. In this particular case, the patient had been followed for 20 years and, while showing some neurologic deficit, there were no signs of recurrence. The tumors in the other 8 cases in this group were meningiomas (3), neurofibromas (2), benign cysts (2), and giant cell tumor (1) which was partially removed from the mid-thoracic region. Of the 2 patients followed for 10 years or more who were not totally cured from the neurologic point of view, one had the giant cell tumor and the other the ependymoma, both of which were partially removed.

Considering that 13 of the total group of patients were paraplegic before surgery, the prognosis in these cases should not be considered hopeless by any means. However, the postoperative result will depend to a great extent on an early diagnosis followed by prompt surgery. And finally, even with early diagnosis and expert surgery an ultimate cure cannot be expected unless the tumor is of benign type.

SUMMARY

1. We have briefly reviewed our series of 30 tumors affecting the spinal cord in children of 15 years and younger.

2. Recovery from a complete paraplegia of either the spastic or flaccid type is rare; and in this series only 1 of 13 paraplegics recovered voluntary movement after surgery.

3. The onset is often obscure and of extremely short duration in children, because of their progressive growth and frequent history of trauma in this age group.

4. The predominant neurologic sign in our series was a sensory level to pin-prick (60 per cent) while pain in the back at the level of the lesion was the commonest complaint (47 per cent).

5. The operative mortality was 6.7 per cent, and a 10-year follow-up study showed that 30 per cent of the patients were alive and well.

6. Sixty per cent of the tumors were benign and of those patients who were classified in the group of 10-year cures, all but 1 had benign tumors.

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COMMUNICATING HYDROCEPHALUS FROM SUBARACHNOID BLEEDING*

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THE relationship of subarachnoid bleeding to subsequent hydrocephalus was first suggested by Bagley almost thirty years ago.[3–5] From clinical and experimental studies of subarachnoid bleeding, he concluded absorption of some blood always takes place, but this is associated with meningeal thickening which may cause a block in the channels through which the fluid is returned to the blood stream. His experimental studies in dogs showed that ventricular enlargement did occur in 30 per cent of puppies after blood was injected into the cisterna magna. In both dog and man, he demonstrated meningeal thickening wherever blood came in contact with the meninges, even in the basal cisterns. He did not actually demonstrate a communicating hydrocephalus, though he did show that intermittent spinal fluid drainage produced clinical improvement.

Although hydrocephalus following subarachnoid bleeding has been described previously,[1,12] a progressive obstruction in the cerebrospinal fluid pathways of the cisterns at the base of the brain which results in severe crippling neurological deficits, and impending death, has not been widely recognized.[12] Strauss *et al.*[14] found 4 "incidental" cases of hydrocephalus without ventricular obstruction at necropsy in their first 13 cases of subarachnoid hemorrhage from aneurysms. Sweet[16] reported 2 cases in 1941 following aneursymal rupture, neither recognized antemortem, and reviewed 6 other similar cases found in the literature. Strain and Perlmutter[12] were the first to designate the point of obstruction in a similar case and successfully treated their patient. Such a hydrocephalus can also follow subarachnoid bleeding from trauma, for Penfield and Cone[9] pointed out in 1943 that "mild degrees of hydrocephalus may be produced by extensive bleeding into the subarachnoid spaces, which tends to block absorption of cerebrospinal fluid for some time after the accident."

Communicating hydrocephalus as a cause of progressing neurological deficits after subarachnoid hemorrhage appears to occur more frequently than the literature would indicate. During the past 3 years, 10 such cases in our series have been recognized because of progressive neurological deficits. A larger group showed mild ventricular enlargement not associated with significant neurological dysfunction. These 10 cases fall into three

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groups based on the etiology of the subarachnoid hemorrhage, namely 1) operative hemorrhage, 2) rupture of intracranial aneurysm and 3) head trauma. Brief clinical summaries of the cases in each group will be presented in sufficient detail to a) demonstrate the characteristic variability of signs and symptoms produced by this type of communicating hydrocephalus; b) show the need for pneumoencephalography at an early date in establishing the diagnosis; c) emphasize that profound and confusing neurological deficits associated with the hydrocephalus may be completely reversed by early treatment.

CASE REPORTS

I. SUBARACHNOID BLEEDING AT OPERATION

Case 1. M.D., a 60-year-old white female. Diagnosis: Intracranial neurofibroma left vagus nerve.

1st Hospitalization, Jan. 18, 1954–Mar. 26, 1954.

History. Hoarseness, and weakness of left shoulder and neck for 6 months with increasing left occipital headache. Weakness and inaccuracy of left hand for 3 months.

Examination. Paralysis of left vocal cord, paresis and atrophy of left shoulder and muscles of the neck, bilateral nystagmus, weakness of left tongue, hyperactive reflexes of upper extremity on the left with ataxia, and an equivocal right Babinski sign were present. Spinal fluid pressure was 150 mm.; protein was 290 mg. per cent. Roentgenograms of skull were normal.

Operation, Jan. 27, 1954. Ventriculography (Fig. 1A) and suboccipital craniectomy with resection of a neurofibroma of the left 10th nerve. At operation, done in the face-down position, a large artery in the tumor capsule bled profusely before it was clipped and blood spurted into and filled the basal cisterns anterior to the brain stem.

Course. Postoperatively, an aseptic meningitis was present for 10 days, secondary to break-down of the blood clot in the cisterns. The cerebrospinal fluid was xanthochromic and contained 2500 white blood cells; protein was 1400 mg. per cent. She improved and was asymptomatic from the 11th day until 6 weeks postoperative.

On Mar. 2, 1954, cerebrospinal fluid pressure was 100 mm. The fluid contained no cells; protein was 128 mg. per cent. On Mar. 6, 1954 there was onset of severe vertigo, nausea, and vomiting associated with the slightest motion of the head. She became completely incapacitated and lost 16 pounds in 10 days because of vomiting and inability to eat. High doses of Dramamine were ineffective.

On Mar. 16, 1954, pneumoencephalography (Fig. 1B) showed definite communicating hydrocephalus with enlarged ventricles as compared to the pre-operative ventriculogram. Spinal fluid pressure was 290 mm.

On Mar. 17, 1954, a right external ventriculomastoidostomy was performed with rapid and complete relief of vestibular irritability.

2nd Admission, Aug. 16, 1954. She had remained asymptomatic. Admission was to determine if the shunt could be terminated.

Examination. Paralysis of the left vocal cord and paresis of the left neck and shoulder persisted. The ventriculomastoidostomy was functioning well.

Course. On Aug. 22, 1954, pneumoencephalography (Fig. 1C) showed return of

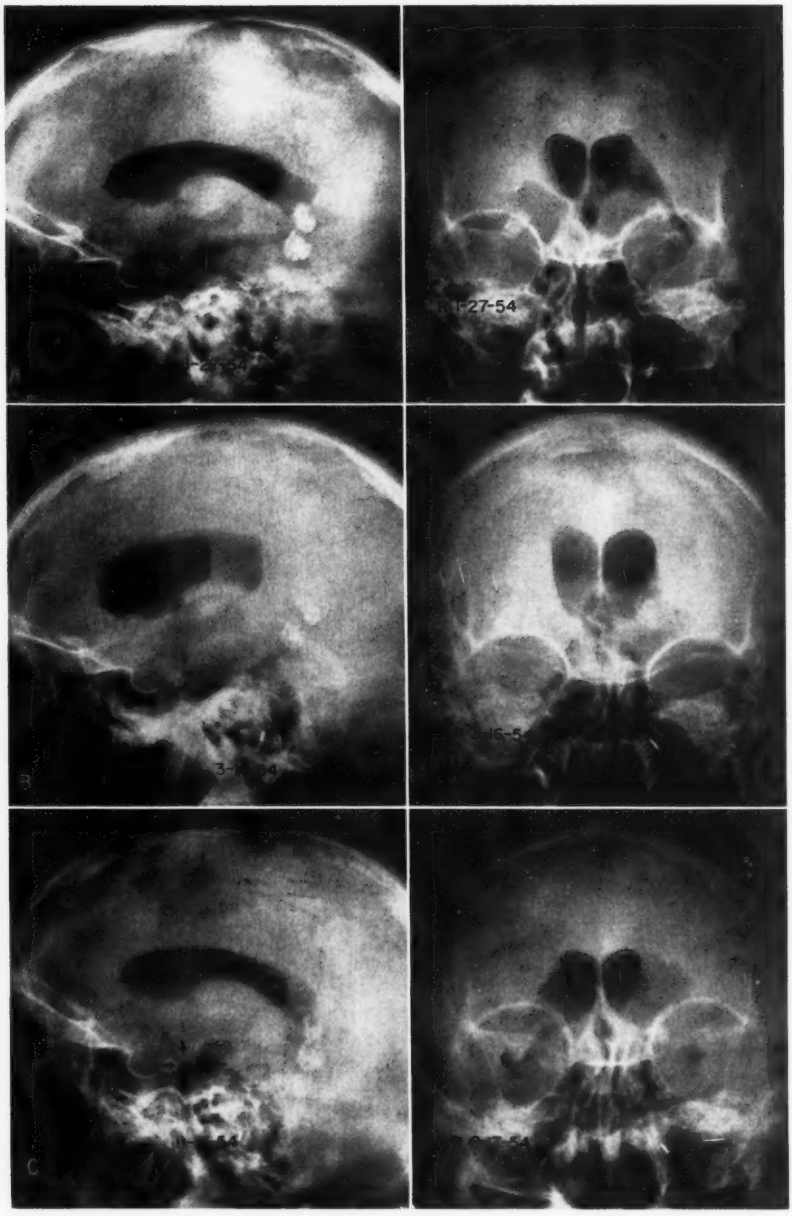

FIG. 1. *Case 1* (left vagus neurofibroma, intracranial; operative hemorrhage). Serial air studies, anteroposterior and lateral ("brow-up") views.

(A) Pre-operative ventriculogram, showing ventricles of normal size.

(B) Pneumoencephalogram, 7 weeks after bleeding into basal cisterns, shows moderately dilated ventricles and subarachnoid block at upper cisterna pontis, with no hemispheral subarachnoid oxygen (every effort was made to fill subarachnoid spaces). Silver clip is on vessel that bled at operation.

(C) Pneumoencephalogram, 5 months after ventriculomastoidostomy, shows return of ventricles to previous size, but subarachnoid block to oxygen in basal cisterns remains. Asymptomatic.

the ventricles to normal size, but still no subarachnoid air over the hemispheres. The shunt was not interrupted.

Follow-up. On July 27, 1955, the only neurological deficit was paralysis of the vocal cord. She was otherwise asymptomatic.

Comments. 1. The communicating hydrocephalus developed as a direct result of operative introduction of blood into the basal cisterns, reaching alarming clinical significance at 2 months.

2. Completely incapacitating vestibular hyperactivity occurred secondary to the communicating hydrocephalus.

3. No papilledema was present at any time. The diagnosis was established only by pneumoencephalography.

4. Complete relief of symptoms and signs of the hydrocephalus, including roentgenographic demonstration of reversal of the ventricular enlargement, followed the ventriculomastoid shunt.

Case 2. T.J., 42-year-old white female. Diagnosis: Left acoustic neurinoma.

1st Hospitalization, Dec. 28, 1951–Jan. 25, 1952.

History. She had had left deafness for 3 years. Severe left occipitofrontal headaches, dizziness, veering to the left, blurring of vision on the left, occasional left facial numbness, and episodic vomiting had appeared in the last 3 months.

Examination. There was bilateral nystagmus, greater on left, with bilateral papilledema (2 D.). She had mild left facial weakness, hypesthesia of the left face and a decreased left corneal reflex. She tended to fall to the left and had ataxic movements on the left. Roentgenograms of the skull showed rarefaction of the left petrous ridge.

Operation. On Jan. 4, 1952, a left acoustic neuroma was removed through a left occipital craniotomy. Intracapsular removal of the large cystic tumor was done medially but extracapsular excision was accomplished laterally. Moderate bleeding from the vascular bed of the tumor occurred during the operation with one episode of profuse bleeding from an artery in the capsule.

Course. Postoperative lumbar puncture revealed grossly bloody cerebrospinal fluid with a pressure of 240 mm. The patient's status improved but was complicated by a presumed exacerbation of a previously diagnosed schizophrenia.

2nd Hospitalization, Mar. 4, 1952–Apr. 10, 1952. Headache and bulging decompression had been present 1 week. Her schizophrenia apparently became so marked that she was sent to a state mental hospital on Apr. 10, 1952, where she was confined until May, 1954.

3rd Hospitalization, June 29, 1955–Aug. 5, 1955. She had severe dizziness on motion of the head, recurrent headache, and occasional vomiting. The occipital decompression had been constantly tense for 6 months. Because of the diagnosis of schizophrenia much of her symptomatology had been discounted for over a year.

Examination. Early choked disc bilaterally, nystagmus in all directions to a marked degree, paresis of the left 5th, 7th and 12th nerves and increased tone in the right arm were present. These deficits were variable.

Course. On July 4, 1955, spinal fluid pressure was 240 mm.; protein was 346 mg. per cent.

On July 13, 1955, combined ventriculogram-pneumoencephalogram indicated a communicating hydrocephalus (Fig. 2) with very large basal cisterns invaginating

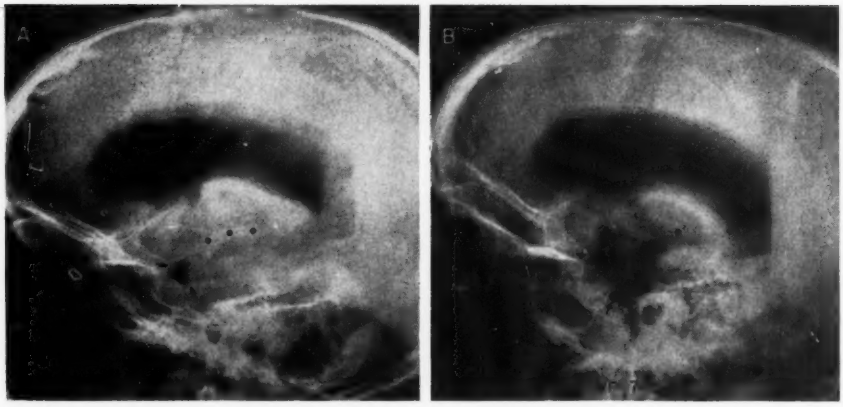

FIG. 2. *Case 2* (left acoustic neurinoma; operative hemorrhage). Postoperative air studies, lateral "brow up" views.

(A) Ventriculogram shows dilated ventricles, including the 4th, with an oxygen bubble in the cisterna chiasmatica (arrows) and elevation of the floor of the 3rd ventricle (dots).

(B) Additional oxygen by pneumoencephalogram shows distended basal cisterns (cisterna pontis, chiasmaticus, and interpeduncularis) invaginating the floor of 3rd ventricle. There is no subarachnoid air beyond these cisterns.

the floor of the 3rd ventricle. There was no convincing evidence of recurrent tumor.

On July 20, 1955, a left ventriculomastoid shunt caused immediate improvement in vision, loss of the dizziness and vomiting, and a soft decompression.

4th Hospitalization, Dec. 2, 1955–Feb. 15, 1956. Reappearance of headaches had occurred 1 week previously.

Examination. The left occipital decompression was soft and pulsating. Secondary optic atrophy was present to a minimal degree. Lumbar puncture showed normal pressure.

Course. While in the hospital, spells of "feeling like falling" began. On Jan. 11, 1956, lumbar puncture showed 350 mm. of pressure. The ventriculomastoidostomy was now no longer functioning. Revision of the shunt caused immediate improvement. On Jan. 25, 1956, spinal fluid pressure was normal. She was last seen on Feb. 15, 1956, at which time she had no complaints, was doing well, and no further neurological deficits appeared.

Comments. 1. The hydrocephalus apparently developed as a result of the blood that escaped into the basal cisterns at the time of operation. Symptoms of the communicating hydrocephalus associated with a bulging of the operative decompression were present 2 months after surgery, though their actual significance was apparently completely overshadowed by her severe schizophrenia for over 2 years. The operative decompression probably was a most helpful safety factor.

2. Progressive headache, vertigo with nausea and vomiting, and visual blurring occurred secondary to the communicating hydrocephalus.

3. Papilledema did appear as a result of the communicating hydrocephalus. The true diagnosis was established only by a combined ventriculo-

gram-pneumoencephalogram. The invagination of the floor of the 3rd ventricle by the distended huge basal cisterns was striking.

4. Prompt relief of the presenting symptoms and signs followed external ventriculomastoidostomy.

II. SUBARACHNOID BLEEDING FROM INTRACRANIAL ANEURYSMS

Case 3. D.R., 27-year-old white female. Diagnosis: Intracranial aneurysm, right internal carotid artery.

History. The patient was found stuporous in her car following an automobile accident.

Examination. A bruise of the right frontal scalp was present with ecchymosis of the right upper eyelid. The fundus was normal on the left. She was semicomatose and her speech was almost dysarthric. There was mild nuchal rigidity. Lumbar puncture showed a pressure of 400 mm. of cerebrospinal fluid, which was grossly bloody.

Course. On May 15, 1955, lumbar puncture showed a pressure of 110 mm.; the spinal fluid was still grossly bloody. Improvement was satisfactory on bed rest.

2nd Hospitalization, May 30, 1955–Aug. 8, 1955. Severe frontal headache had developed 1 week prior to admission, associated with staggering gait, confusion, and blurring of vision. She apparently was having auditory hallucinations.

Examination. The patient was confused, disoriented, and semicomatose. The left pupil was slightly larger than the right. There was right lower facial weakness and left lateral gaze was not sustained. Questionable early edema of the optic discs was present bilaterally.

Course. Ventricular tap yielded bloody fluid under 400 mm. of pressure. Ventriculography on May 30, 1955 showed tremendous dilatation of the ventricular system. The release of the ventricular fluid caused a dramatic alleviation of her semicoma. Bilateral exploratory twist drill holes over the posterior fossa showed no posterior fossa subdural hematoma.

On May 31, 1955, ventricular pressure was 390 mm. Release of the ventricular fluid again awakened the patient from a semicomatose state. In each instance, such arousal resulted in reappearance of auditory hallucinations.

On June 1, 1955, continuous ventricular drainage for 5 days caused marked improvement. The drainage was then discontinued.

On June 12, 1955, semicoma had reappeared. Lumbar puncture revealed a pressure of 600 mm. of cerebrospinal fluid. Combined lumbar and ventricular punctures showed good communication.

On June 23, 1955, bilateral arteriography demonstrated intracranial aneurysm of the internal carotid artery on the right associated with a sweeping course of the anterior cerebral arteries.

From July 1, 1955 to July 5, 1955, daily lumbar punctures (600 mm. pressure) caused marked improvement from semicoma to almost normal alertness lasting 10 to 12 hours.

On July 5, 1955, pneumoencephalography (Fig. 3) showed marked communicating hydrocephalus.

On July 7, 1955, a right ventriculomastoidostomy caused immediate clinical improvement with normal speech, awareness of her surroundings, and the realization that her vision was markedly impaired. Bilateral early optic atrophy was present which appeared at this time to be primary optic atrophy.

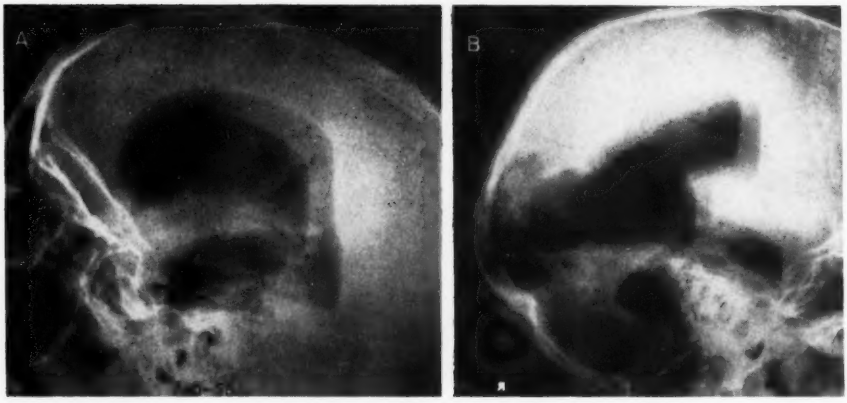

FIG. 3. *Case 3* (aneurysm of internal carotid artery, right). Pneumoencephalogram 7 weeks after bleeding.

(A) Lateral "brow up" view, showing advanced ventricular dilatation and block to subarachnoid oxygen at cisterna chiasmaticus and interpeduncularis (arrows) with no hemispheral subarachnoid oxygen.

(B) "Brow down" lateral view. The enlarged 4th ventricle was present in all cases.

On July 26, 1955, minimal gross vision to light was present on the right only. She continued to improve satisfactorily otherwise.

3rd Hospitalization, Sept. 25, 1955–Oct. 24, 1955. A lumbar puncture elsewhere on Sept. 19, 1955 had shown 400 mm. of pressure. She had become lethargic, had been hallucinating again and the severe bilateral optic atrophy was unimproved.

Examination and Course. The patient showed moderate lethargy and was having episodes of auditory hallucinations. The shunt was not functioning. On Oct. 12, 1955 revision of the ventriculomastoidostomy resulted in almost immediate improvement in awareness and loss of hallucinations. Postoperative spinal fluid pressure was 80.

4th Hospitalization, Feb. 23, 1956–Mar. 10, 1956. Mild reduction in psychomotor activity and increased lethargy had appeared 3 weeks prior to admission, associated with elevation of spinal fluid pressure to 400 mm. There was persistence of 95 per cent of her amaurosis.

Examination and Course. Primary optic atrophy and moderate lethargy were present. Spinal fluid pressure was 460 mm. On Mar. 2, 1956, revision of mastoidostomy resulted in immediate improvement and normal cerebrospinal fluid pressure.

Comments. 1. The communicating hydrocephalus developed as a result of two episodes of bleeding into the basal cisterns from the aneurysm of the right internal carotid artery. Symptomatic hydrocephalus was present 2 weeks after the initial hemorrhage.

2. Headache, confusion, semicoma, and auditory hallucinations were the clinical manifestations of the progressing hydrocephalus, repeatedly relieved by lumbar puncture and on three occasions by ventriculomastoid shunt.

3. No frank papilledema was present at any time, but early loss of vision of maximum degree was present 5 weeks after the second hemorrhage

associated with development of primary optic atrophy. The true diagnosis of communicating hydrocephalus was not established by ventriculography or arteriography, but was firmly established by pneumoencephalography.

4. Prompt relief of the incapacitating signs and symptoms of the hydrocephalus was achieved by external ventriculomastoidostomy.

Case 4. M.J.R., 26-year-old white female. Diagnosis: Aneurysm, basilar artery.

1st Hospitalization, July 19, 1955.

History. Four days previously, the patient was unconscious on removal from an automobile accident. No focal neurological deficits were reported. Lumbar puncture had showed grossly bloody cerebrospinal fluid with a pressure of 220 mm. She had regained consciousness after 10 hours, but had remained disoriented.

Examination. The patient complained of severe headache and was disoriented. Left 6th nerve palsy, positive Babinski sign on the right, and nuchal rigidity were present. A moderately severe brain stem contusion was presumed to be secondary to head trauma. Roentgenograms of the skull were normal.

Course. On July 31, 1955, vomiting episodes lasted for 2 days. Gradual improvement occurred on bed rest.

On Aug. 2, 1955, the cerebrospinal fluid pressure was 180 and the fluid was xanthochromic.

On Aug. 6, 1955, sudden coma occurred with tonic left adversion. The cerebrospinal fluid was again grossly bloody, under a pressure of 360 mm. Coma for 6 hours was followed by gradual improvement.

On Aug. 9, 1955, sudden coma occurred again with grossly bloody cerebrospinal fluid under a pressure of 600 mm. Complete right 6th nerve palsy, poor responses to pain in the arms and occasional decerebrate response in the right arm were present. Positive Babinski sign was present bilaterally. A diagnosis of intracranial aneurysm was made.

By Aug. 22, 1955, gradual improvement had occurred, but the patient was dull, lethargic, and expressionless. Bilateral 6th nerve palsy was now present, and there was paresis of superior-inferior gaze on the right. The Babinski sign was present on the right only. Severe headache and a rigid stiff neck persisted.

On Aug. 26, 1955, bilateral carotid arteriography did not reveal the cause of bleeding, but sweeping anterior cerebral arteries indicated large ventricles.

On Sept. 1, 1955, vertebral arteriography showed the basilar artery to be deformed at its upper portion, suggestive of a malformation or an incompletely filled aneurysm.

On Sept. 2, 1955, early papilledema and tonic head-neck adversion to the left were present. Rapid progression from moderate lethargy to complete coma and nearly terminal state occurred that night. An emergency ventriculomastoidostomy was done under local anesthesia. The intraventricular pressure measured 700 mm. of clear cerebrospinal fluid. Immediate dramatic improvement followed this procedure with rapid change from deep coma and flaccidity to a state of responsiveness with active reflexes.

By Sept. 12, 1955, steady improvement had occurred. The patient was alert, oriented, and totally amnesic for the prior 2 months. A very flat affect and lack of psychomotor activity were present with only a minimal right 6th nerve palsy persisting. The cerebrospinal fluid pressure was 100 mm. and the fluid itself was normal.

On Sept. 26, 1955, pneumoencephalography demonstrated a typical communi-

cating hydrocephalus of a moderate degree with the block to air at the basal cisterns (as in Fig. 1).

On Oct. 18, 1955, her neurological findings were normal.

Comments. 1. The communicating hydrocephalus resulted from subarachnoid bleeding caused by a basilar aneurysm. Three episodes of bleeding occurred. The hydrocephalus reached nearly fatal proportions 2 months after the first hemorrhage.

2. A fatality from the complicating communicating hydrocephalus was narrowly avoided. Headache and disorientation, followed by lethargy and coma associated with bilateral 6th nerve palsy and pyramidal signs were caused by the hydrocephalus. The other brain stem signs were presumably secondary to the vascular lesion itself.

3. Early papilledema was present. Diagnosis of the hydrocephalus was suggested by carotid arteriography, but was firmly established only by pneumoencephalography after emergency treatment had been done.

4. Rapid and dramatic reversal of the nearly terminal state was achieved by the external ventriculomastoidostomy. Recovery to a normal neurological status was achieved.

Case 5. W.M., 66-year-old white male. Diagnosis: Basilar artery aneurysm.

First Hospitalization, Nov. 15, 1953–Nov. 25, 1953.

History. Collapse and coma occurred 3 weeks prior to admission preceded by severe occipital headache. Initially, there were frequent emesis, ptosis of the right eyelid, and grossly bloody cerebrospinal fluid. Coma persisted for 4 hours. Six days later, after almost complete neurological recovery with clearing of the cerebrospinal fluid, a second subarachnoid hemorrhage occurred, associated with stiff neck, right ptosis, dilated right pupil, hyperactive left tendon reflexes, and bilateral Babinski sign. The cerebrospinal fluid was again grossly bloody with a pressure of 600 mm. Semicoma had persisted during the following 10 days.

Examination. The patient was comatose with severe nuchal rigidity and a dilated, fixed right pupil with right ptosis. There was no response to speech or painful stimuli. Increased tone was present in the lower extremities with bilateral extensor plantar responses and hyperactive tendon jerks.

Course. On Nov. 20, 1953, bilateral carotid arteriography showed a small aneurysm on the right middle cerebral artery (considered to be incidental) and a sweeping course of the anterior cerebral arteries. Diagnosis of probable communicating hydrocephalus secondary to recurrent bleeding into the basal cisterns was made.

On Nov. 25, 1953, vertebral arteriography was scheduled, but a third subarachnoid hemorrhage was fatal.

Postmortem Examination. A large aneurysm of the basilar artery at its bifurcation into the posterior cerebral artery, measuring 1×2.5 cm., was present. The basal cisterns were obliterated with organizing and fresh clot. The entire ventricular system, including the 4th ventricle, was grossly dilated. The aneurysm of the right middle cerebral artery was unruptured.

Comments. 1. The communicating hydrocephalus resulted from massive bleeding into the basal cisterns from a large aneurysm of the upper portion

of the basilar artery. The time from the first hemorrhage until enlarged ventricles were shown by arteriography was 4 weeks.

2. No signs and symptoms can be attributed to the hydrocephalus.

3. No papilledema was present. The diagnosis was firmly established only at postmortem examination.

4. No operative procedure was done to treat the hydrocephalus.

Case 6. V.H., 28-year-old white male. Diagnosis: Aneurysm of left anterior cerebral artery.

First Hospitalization, Apr. 14, 1953–Jan. 10, 1954.

History. The patient had a severe personality disturbance and "blackout" spells. While under psychiatric investigation, a subarachnoid hemorrhage occurred on June 2, 1953.

Examination. On June 15, 1953, mild nuchal rigidity was present associated with an equivocal right Babinski sign. On June 16, 1953, carotid arteriography showed a large aneurysm of the left anterior cerebral artery (Fig. 4A).

Course. On June 22, 1953, a second subarachnoid hemorrhage occurred and the patient rapidly deteriorated. Craniotomy was performed and the neck of the aneurysm was successfully clipped with a large tantalum clip. Postoperative improvement was slow but steady over a 3-week period.

On July 15, 1953, the personality disturbance reappeared. The patient was transferred to the Psychiatry Service.

On Sept. 11, 1953, there had been 2 weeks of headache, steady deterioration, and episodes of catatonic-like spells lasting 10 to 18 hours. Postoperative arteriography showed non-filling of the aneurysm and a sweeping course of the anterior cerebral arteries (Fig. 4B). Spinal fluid pressure was 90 mm.; the fluid was normal.

On Oct 29, 1953, pneumoencephalography demonstrated marked communicating hydrocephalus with the block to oxygen at the basal cisterns. The patient had

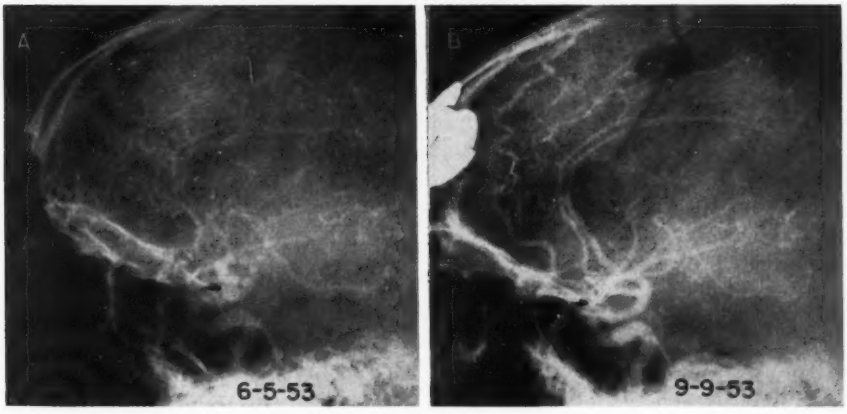

FIG. 4. *Case 6* (aneurysm of anterior cerebral artery, left).
(A) Left carotid arteriogram 2 weeks after bleeding shows aneurysm (arrow).
(B) Ten weeks after craniotomy, arteriogram shows advancing ventricular enlargement and obliteration of aneurysm by clip (arrow).

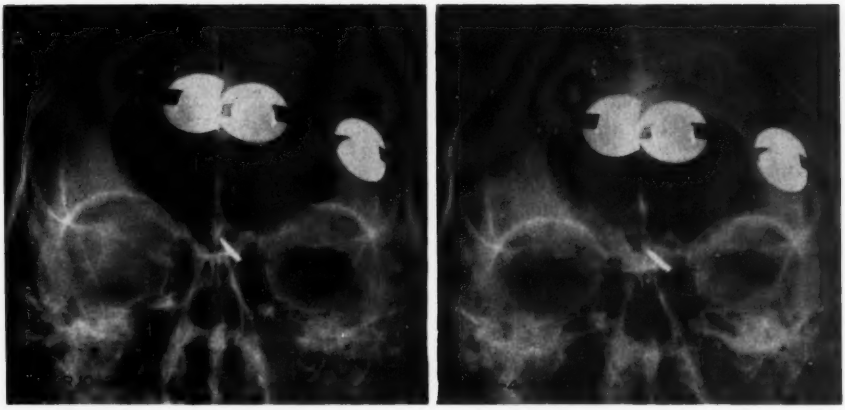

FIG. 5. *Case 6* (aneurysm of anterior cerebral artery, left).

(A) Pneumoencephalogram 4 months after second bleeding from aneurysm shows extensive ventricular enlargement from communicating hydrocephalus with *normal* spinal fluid pressure; patient catatonic.

(B) Two months after right external ventriculomastoidostomy, size of ventricles was reduced, spinal fluid pressure was normal, and patient was neurologically normal.

progressed to a catatonic-like state with mask-like facies, complete and total disorientation, and bilateral pyramidal and extrapyramidal signs of a severe degree. He was barely vegetating.

On Nov. 17, 1953, right ventriculomastoidostomy was done. The intraventricular pressure was less than 50 mm. The neurological recovery during the next 4 days was startling. He became practically normal objectively, but memory deficits for the previous 2 months persisted.

On Jan. 4, 1954, repeat pneumoencephalography showed definite decrease in the size of the ventricles though the communicating hydrocephalus persisted (Fig. 5).

Follow-up. On July 5, 1955, the patient was normal neurologically and had returned to work. The ventriculomastoidostomy was still functioning intermittently.

Comments. 1. The communicating hydrocephalus developed as a result of two episodes of bleeding into the basal cisterns from rupture of the anterior cerebral aneurysm. Evidence of this hydrocephalus was present 10 weeks after the last hemorrhage.

2. Minimal headache, confusion, lethargy, catatonic-like state, and severe bilateral pyramidal and extrapyramidal signs were precipitated by the communicating hydrocephalus.

3. *No increase* in intracranial pressure was recorded at any time. The diagnosis was first indicated by the repeat arteriography, and confirmed by pneumoencephalography.

4. Dramatic reversal of a severely depressed, catatonic-like state was achieved by the ventriculomastoidostomy. Postoperative reduction in size of the ventricles was demonstrated by air studies.

Case 7. J.D.M., 22-year-old white male. Diagnosis: Aneurysm of right anterior cerebral artery.

1st Hospitalization, May 26, 1954–June 6, 1954.

History. Throbbing bitemporal headache of increasing severity had been present for 6 months. The patient had had a subarachnoid hemorrhage without focal neurological deficits 18 months prior to admission. Carotid arteriography (February 1953) showed an aneurysm of the right anterior cerebral artery.

Examination. There was no neurological abnormality. Roentgenograms of the skull were normal and serial electroencephalography indicated a deep subcortical lesion. Spinal fluid pressure was normal, and the fluid was normal.

Course. On May 27, 1954, bilateral carotid arteriography showed a large aneurysm of the right anterior cerebral artery.

2nd Hospitalization, Oct. 13, 1954–Dec. 5, 1954. The patient had had two episodes of severe bitemporal headache associated with stiff neck during the interval since discharge. No neurological deficits had occurred. Both episodes lasted 3 days.

Examination. There were no neurological abnormalities.

Course. On Oct. 5, 1954, repeat carotid arteriography again showed good filling of the right anterior cerebral aneurysm. The aneurysm had enlarged since the last arteriogram. The anterior cerebral artery followed a sweeping course.

On Oct. 22, 1954, at craniotomy, the right anterior cerebral artery was occluded with a silver clip just proximal to the aneurysm. The postoperative course was very precarious for 8 days, followed by gradual improvement. He was normal neurologically 6 weeks after operation.

3rd Hospitalization, Feb. 1, 1955–Feb. 12, 1955. No significant events had occurred in the interval.

Examination. Neurological findings were still normal.

Course. On Feb. 4, 1955, repeat carotid arteriography showed no filling of the aneurysm on bilateral injection. The sweeping course of the anterior cerebral arteries had not changed. A diagnosis of arrested communicating hydrocephalus was made. No active treatment was warranted.

Comments. 1. The hydrocephalus developed as a result of bleeding into the basal cisterns from rupture of the anterior cerebral aneurysm. At least three such hemorrhages had occurred.

2. No symptoms could be attributed to the arrested hydrocephalus except possibly the mild headaches.

3. No evidence of increased intracranial pressure or papilledema was present at any time. Hydrocephalus was obvious from the arteriograms, but final proof that it was a communicating hydrocephalus by pneumoencephalography was not done.

4. Lack of clinical symptoms together with evidence of no progression by serial arteriography indicated the hydrocephalus was arrested and no treatment was warranted.

III. SUBARACHNOID BLEEDING FROM HEAD TRAUMA

Case 8. S.L.S., 4-year-old white female. Diagnosis: Severe cerebral contusion.

1st Hospitalization, Jan. 15, 1954–Jan. 20, 1954.

History. The patient had fallen from a moving car on Dec. 25, 1953. She had remained comatose since first seen. A basal skull fracture extending down into the foramen magnum was present. Antibiotics, nasal oxygen, and supportive care had been given for 3 weeks.

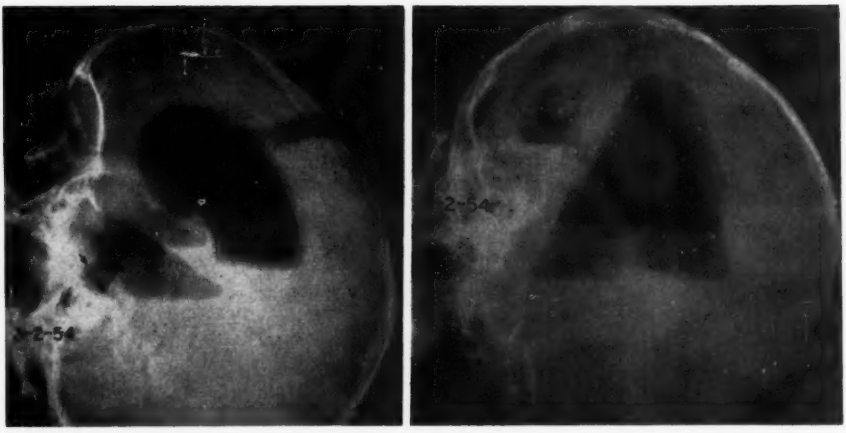

FIG. 6. *Case 8* (head injury with subarachnoid bleeding). Pneumoencephalogram 9 weeks after injury, "brow up" and "brow down" lateral views. There is advanced ventricular enlargement, including 4th ventricle, distended cisterna pontis and interpeduncularis (arrows), with no hemispheral subarachnoid oxygen. Suture lines are separated.

Examination. The child was comatose, but would open her eyes on painful stimulation, without ocular fixation. Bilateral 6th nerve palsy, peripheral right 7th nerve palsy, and right facial hypalgesia were present. Reflexes of decerebrate type, in response to painful stimuli, were present in the left arm and leg. A right Babinski sign was present. Lumbar puncture yielded normal spinal fluid under normal pressure. Vitreous hemorrhages on the right caused blindness, but the left pupillary reflexes were normal. She was alert, moving about well in bed, and eating at discharge.

2nd Hospitalization, Mar. 1, 1954–Mar. 12, 1954. Three weeks of increasing lethargy and occasional vomiting had occurred. Ten days prior to admission, roentgenograms of the skull showed separation of the sutures. Swelling over the site of the fracture at the foramen magnum had appeared.

Examination. The patient was very drowsy, and would say "no" and "mama" only. Bilateral 6th nerve palsy was present. Bilateral amaurosis was present with extensive vitreous opacities in the right eye and optic atrophy on the left. There was increased extensor tone in the legs with bilateral Babinski sign.

Course. On Mar. 2, 1954, pneumoencephalography showed extensive communicating hydrocephalus (Fig. 6) with ballooned and obstructed basal cisterns with no subarachnoid air over the hemispheres.

On Mar. 5, 1954, right external ventriculomastoidostomy caused immediate improvement in consciousness. The occipital decompression became soft and pulsating.

Follow-up. On Nov. 12, 1954, the patient was alert and active. Except for minimal residua of brain stem damage, the major disability was blindness from left optic atrophy, and vitreous opacities in the right eye.

Comments. 1. The communicating hydrocephalus appeared clinically 6 weeks following severe bleeding around the base of the brain from trauma.

2. Confusion and marked lethargy with a nearly terminal appearance were the only signs apparently produced by the hydrocephalus. The rest of

the disabilities appeared to be the result of the original brain stem trauma.

3. High intracranial pressure caused separation of the cranial sutures. No papilledema was seen, though the right fundus was obscured. Pneumoencephalography established the true diagnosis.

4. Rapid reversal of the nearly terminal status occurred following external ventriculomastoidostomy under local anesthesia.

Case 9. R.E.S., 64-year-old white male. Diagnosis: Head trauma with subarachnoid bleeding.

1st Hospitalization, Mar. 5, 1952–June 4, 1952.

History. Eleven years prior to admission, the patient fell off a truck and was knocked unconscious. Spinal fluid was "very bloody." Stiff neck and severe headache were present for several weeks One year later, disturbance in walking and gait had appeared and had been steadily progressive. Mental deterioration was said to have been present for 1 year.

Examination. The patient walked with a broad-based, shuffling, mincing gait. The deep tendon reflexes were definitely hyperactive generally.

Course. Gastro-intestinal and psychiatric evaluations were extensive.

2nd Hospitalization, Aug. 18, 1953–Nov. 1, 1953. There had been progressive difficulty in walking and increasingly poor intellectual function.

Examination. Extremely poor intellectual function, preoccupation, distractability, and loss of memory were prominent. Weakness and spasticity of the lower extremities were present with ataxia, bilateral ankle clonus, bilateral Babinski sign, bilateral palmomental reflex, and positive snouting. His gait was slow, shuffling and propulsive.

Course. Spinal tap showed a pressure of 100. The cerebrospinal fluid protein was 50 mg. per cent.

On Sept. 24, 1953, pneumoencephalography showed advanced communicating hydrocephalus (Fig. 7) with a block to oxygen at the basal cisterns. Daily lumbar punctures did not improve his deteriorated status.

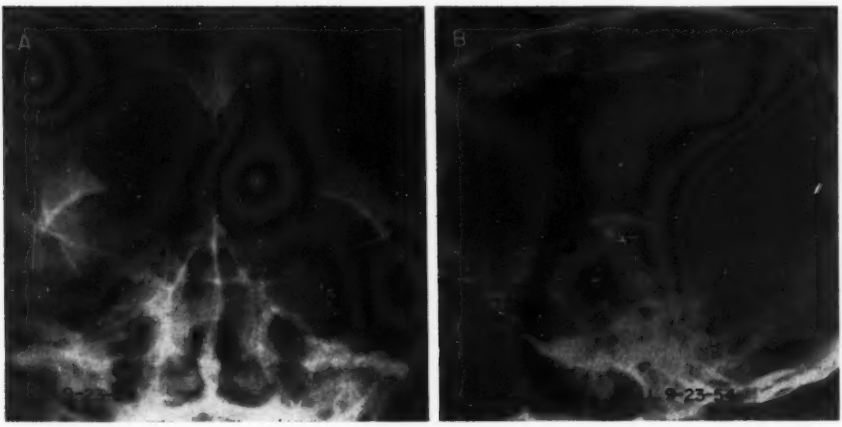

FIG. 7. *Case 9* (multiple head injuries with subarachnoid bleeding, old). Pneumoencephalogram in anteroposterior and lateral views, showing tremendous ventricular enlargement in arrested communicating hydrocephalus with extensive cerebral atrophy; prolonged, unamenable to therapy.

3rd Hospitalization, Jan. 15, 1954–May 3, 1954. There had been slow deterioration to complete dependency.

Examination. There was accentuation of previous findings.

Course. On Jan. 18, 1954, repeat pneumoencephalography showed findings identical to those of the previous study with no convincing evidence of progression.

On Jan. 22, 1954, external ventriculomastoidostomy was unsuccessful since the pressure gradient was unfavorable. Intraventricular pressure was less than 80 mm.

Follow-up. On July 20, 1955, slow, steady, mental deterioration had progressed with little change in the spasticity and ataxia of the lower extremities. He was a completely dependent neurological cripple.

Case 10. C.D., 50-year-old white male. Diagnosis: Recurrent head trauma with subarachnoid bleeding.

1st Hospitalization, Dec. 22, 1955.

History. Generalized seizures and coma had occurred in the city jail. He had been admitted to the hospital on numerous previous occasions with head injuries suffered in 1937, 1938, 1939, and a severe one in 1955. These head injuries had not caused focal neurological deficits. Lumbar punctures had been done only on the last two admissions, both showing bloody cerebrospinal fluid. Chronic alcoholism partially obscured his past history, but apparently multiple other head injuries had not required hospitalization.

Examination. Confusion and disorientation were prominent without focal neurological deficits.

Course. Lumbar puncture revealed normal pressure and normal cerebrospinal fluid.

On Dec. 27, 1955, pneumoencephalography showed tremendous communicating hydrocephalus (as in Fig. 7). Sufficient improvement in confusion and disorientation allowed discharge subsequently, with seizures controlled.

On Jan. 17, 1956, repeat pneumoencephalography showed no significant change in the hydrocephalus. Daily lumbar punctures gave no improvement. He was transferred to a hospital for chronic care, where his confusion and mild disorientation persisted.

Comments. 1. The logical cause for the communicating hydrocephalus in both Cases 9 and 10 was the obvious head injuries and subarachnoid bleeding.

2. The advanced cerebral deterioration was probably a secondary result of severe communicating hydrocephalus of long standing.

3. No evidence of increased intracranial pressure was observed at this late stage of the hydrocephalus. Serial pneumoencephalography showed no evidence of progression of the communicating hydrocephalus.

4. Lack of response to lumbar punctures and no radiological evidence of progression indicated an irreversible hydrocephalus. Ventriculomastoidostomy was technically inadvisable and not warranted since the cerebral damage already present appeared to be chronic and permanent.

DISCUSSION

Communicating hydrocephalus may result when an inflammatory reaction is produced in the basal cisterns by introduction of a foreign sub-

stance, including infection,[6] certain chemicals[11,15] and blood.[2–5,9,12,16] The only common factor in the 10 cases of this series was the subarachnoid bleeding. In addition, the source of this bleeding in all 8 cases in which the causative lesion was positively identified was located on the ventral surface of the brain in an ideal position to bleed directly into the cisterns and pack them with blood. The subarachnoid block in all instances proved to be in the basal cisterns or their immediate periphery. The aneurysms were located on proximal portions of the anterior cerebral artery, on the internal carotid just below the bifurcation, or on the upper part of the basilar artery. Operative hemorrhage occurred from capsular vessels of benign tumors located anterolateral to the brain stem. The head injuries had produced marked contusion of the upper portion of the brain stem. The assumption that the hydrocephalus is the result of an adhesive arachnoiditis of the basal leptomeninges is supported by previous experimental[2,3] and clinical[4,5,9,12,14,16] observations as well.

The time after hemorrhage before the communicating hydrocephalus was recognized or produced symptoms was variable. Table 1 shows this time to vary from 2 weeks up to 12 weeks. There appeared to be no relation of this interval to the number of hemorrhages that had occurred. However, there does appear to be a relationship between the time of onset of symptoms and the degree of intracranial hypertension. Apparently, those with high intracranial pressure show symptoms referable to the hydrocephalus earlier, as in Cases 3, 4, 5 and 8 (Table 1). In 3 of these same cases (Cases 3, 4 and 8) the patients came very close to death because of the hydrocephalus and were saved only by the ventriculomastoid shunt. The other surviving patients had lower pressure and were not as critically ill, though all were severely crippled neurologically. Thus it would appear that the hydrocephalus resulting from subarachnoid bleeding may be a progressive lesion, and a massive subarachnoid block will be reflected by early,

TABLE 1

Time interval between hemorrhage and symptoms of hydrocephalus, and relationship of onset of symptoms to degree of intracranial hypertension

Case No.	Number of Hemorrhages	Time after Hemorrhage Symptoms Appeared	Pressure, Spinal or Ventricular
1	1	7 weeks	290
2	1	12 weeks	240
3	2	2 weeks	350–400
4	3	8 weeks	700
5	3 (death)	4 weeks	600
6	2	12 weeks	*50*
7	3	? (arrested)	100
8	1	8 weeks	450
9	unknown, but several	unknown, present several years	110
10	unknown, but several	unknown, present several years	100

significant elevation of intracranial pressure. This may be followed by a fatal outcome unless adequate treatment is given.

The role of increased intracranial pressure in the symptomatology and ventricular enlargement of communicating hydrocephalus is not entirely clear. When communicating hydrocephalus was discovered in Case 6, normal intracranial pressure was present. Even so his severe, crippling neurological deficits were dramatically reversed by the ventriculomastoidostomy and the enlarged ventricles showed definite reduction in size by roentgen ray after the shunt (Fig. 6). It is therefore impossible to attribute all the neurological deficits to increased intracranial pressure alone. This particular case indicated, moreover, that even with normal intracranial pressure, the deficits attributable to the hydrocephalus can still be reversed by a shunt operation. However, these changes cease to be reversible and become permanent after a period of time, as shown by Cases 9 and 10. In these two instances the hydrocephalus had been present for a period of years and the neurological deficits could not be reversed by cerebrospinal fluid shunts, or repeated lumbar punctures.

TABLE 2

Signs and symptoms associated with the communicating hydrocephalus

Signs and Symptoms	Etiology of Subarachnoid Hemorrhage								
	Operative		Aneurysm					Trauma	
	Case 1	Case 2	Case 3 Int. Carot.	Case 4 Basilar	Case 5* Basilar	Case 6 Ant. Cereb.	Case 7 Ant. Cereb.	Case 8	Cases 9 & 10†
Increased pressure	x	x	x	x	x			x	
Headache	x	x	x	x	x	x	x		
Vertigo, nausea, and vomiting	x	x							
Confusion to disorientation			x	x	x	x		x	x
Lethargy, semicoma, or coma	x		x	x	x	x		x	
Hallucinations			x auditory						
Visual blurring		x							
Papilledema		x minimal							
Optic atrophy		x secondary	x primary					x lt. prim.	
IV nerve palsy				x bilat.					
Internuclear palsy				x					
Tonic adversion				x					
Catatonic-like state						x			
Pyramidal signs				x		x			
Extrapyramidal signs						x			

* Died from rerupture of aneurysm.

† Arrested cases.

The clinical signs and symptoms that accompany this delayed hydrocephalus are varied. Table 2 compares the signs and symptoms in this group. There is no constant syndrome caused by the hydrocephalus, though headache was the most common symptom. Increased spinal fluid pressure was present in all rapidly progressing cases. Although the signs and symptoms are variable and possibly confusing, they would all appear to be secondary to dysfunction of basal midline structures, including brain stem, midbrain and ventral diencephalon as well as optic nerves. This may well be related to the fact that the lesions causing the hemorrhage are closely associated with these structures and also the adhesive arachnoiditis resulting from the hemorrhage appears to involve the basal leptomeninges primarily.

Papilledema was not observed in any of the patients with increased intracranial pressure, though suggestive papilledema was present in Case 2. This is explained on two possible bases. The first possibility is a perineural fibrosis of the optic nerves. Griffith *et al.*[8] have shown experimentally that subarachnoid hemorrhage filled the perineural spaces of the optic nerves with blood. The resulting adhesions obliterated this space and so prevented papilledema even in the presence of experimental cerebellar neoplasms. Since cerebrospinal fluid escapes along this perineural space[11,13,15] from the basal cisterns, papilledema can not occur if this space is obliterated. The second possibility is a chiasmatic arachnoiditis which, if sufficiently severe, will likewise prevent papilledema. In either instance, primary optic atrophy may result, as occurred in Cases 3, 4 and possibly Case 8.

In none of the cases presented was the diagnosis of complicating, progressing communicating hydrocephalus made until it was demonstrated by air study. In 3 instances, arteriography demonstrated enlarged ventricles, but pneumoencephalography was necessary in cases showing progression to prove finally the location of the basal subarachnoid block. The obstruction to cerebrospinal fluid circulation is presumably produced through pial-arachnoid adhesions in the basal cisterns. This adhesive arachnoiditis may actually obliterate the cistern, or the adhesions may occur at the periphery of these cisterns, preventing escape of cerebrospinal fluid and causing them to balloon and dilate, invaginating the floor of the 3rd ventricle (Case 2).

The occurrence of this type of progressing hydrocephalus after subarachnoid bleeding may be more common than is generally recognized. Whenever there is bleeding of significant degree into the subarachnoid space, some obstruction of the subarachnoid space results.[3–5,9,16] When this subarachnoid bleeding is of mild degree or involves portions of the subarachnoid space distal to the basal cisterns, ventricular enlargement will ordinarily not occur. However, when a significant hemorrhage is present that involves the basal cisterns, some ventricular enlargement occurs. If the acute clot that forms in the basal cisterns is large enough to cause immediate obstruction, acute elevations of intracranial pressure with subsequent ventricular enlargement will occur, as reported by Goinard *et al.*[7] and as occurred in the current series. Under these circumstances, lumbar puncture (often per-

formed for purely diagnostic purposes) will cause a dramatic relief of signs and symptoms. This clinical observation has been widely recognized, although multiple unsubstantiated explanations for it have been offered. Thus repeated lumbar punctures may well be of therapeutic value in cases of significant subarachnoid bleeding from ruptured aneurysm, head injury or operative hemorrhage.

In the majority of instances of subarachnoid bleeding, any obstruction of cerebrospinal fluid that occurs is present only acutely and does not result in an adhesive arachnoiditis of sufficient degree to produce a delayed, progressive, communicating hydrocephalus (Case 7). However, in a certain number of cases the patient will recover temporarily from the acute obstruction by clot in the basal cisterns. When the clot liquefies, an inflammatory response is evoked with a secondary adhesive arachnoiditis which, after a period of time, may progressively obstruct the cerebrospinal fluid pathways, resulting in the delayed appearance of clinically significant hydrocephalus. Nine of the 10 cases presented fall into this group. In the past, this type of case has usually gone unrecognized, and the signs and symptoms of clinical deterioration have been ascribed to non-specific delayed effects of the original lesions. If this delayed hydrocephalus compensates spontaneously, the patient may survive though severely handicapped.

Once the delayed hydrocephalus is shown to be progressing, urgent treatment may be indicated. In this series, all 6 patients treated by external ventriculomastoidostomy showed dramatic and rapid reversal of their neurological deficits, and in 3 of these cases death was narrowly averted. This procedure is carried out under local anesthesia and consists of inserting a polyethylene catheter into the lateral ventricle through a twist drill hole, the tube being carried subcutaneously to the base of the mastoid, where the distal end is inserted into a tightly fitting twist drill hole into a mastoid air cell. The pressure necessary to drive saline through the eustachian tube is routinely measured by a water manometer and is usually in the range of 80–100 mm. cerebrospinal fluid. The advantages of the procedure are 1) technical simplicity of the procedure; 2) easily performed under local anesthesia; 3) the production of a physiological release valve for escape of cerebrospinal fluid from the ventricles in the presence of increased intraventricular pressure. The first two advantages are particularly applicable in severely ill patients. The presence of a physiological release valve permits flow as long as intracranial pressure is elevated. If intracranial pressure returns to normal and the hydrocephalus becomes arrested, flow will cease and fibrosis within the mastoid will plug the tube. Thus the shunt is self-limiting. If the shunt should cease functioning prematurely, it is easily revised. Although the presence of this valve mechanism in the adult middle ear is a protection to retrograde infection, the occasional complication of meningitis is the major disadvantage that has been encountered.

These observations concerning communicating hydrocephalus in adults

have led us to speculate regarding the genesis of the so-called idiopathic communicating hydrocephalus of infancy. Although pathological studies have not yielded clear concepts regarding etiology, Penfield and Elvidge[10] pointed out in 1932 that an adhesive arachnoiditis was present in approximately one-half of the cases of infantile hydrocephalus studied. However, little attention has been given to subarachnoid bleeding as a possible etiological agent in congenital hydrocephalus, even though Bagley[4] long ago proposed that this may be important. It is generally accepted that 15 per cent of newborn infants have blood in the cerebrospinal fluid, presumably secondary to brain trauma from moulding of the head during birth. Significant bleeding into the basal cisterns could produce a progressive hydrocephalus which would not become clinically obvious for weeks or even months, judging from the data reported in adults. However, discrete information regarding these speculations on the etiology of idiopathic infantile hydrocephalus is lacking and we are presenting this concept only as an hypothesis.

SUMMARY

1) It is proposed that massive subarachnoid bleeding into the basal cisterns can evoke an adhesive arachnoiditis of the basal leptomeninges with a resulting delayed, progressive hydrocephalus. Ten cases are presented.

2) The etiology of the subarachnoid bleeding was operative hemorrhage, ruptured intracranial aneurysms, and cerebral trauma.

3) The delayed, progressive nature of this communicating hydrocephalus producing advancing neurological deficits of a crippling and possibly fatal nature is emphasized. Recognition of the hydrocephalus after hemorrhage varied from 2 to 12 weeks.

4) The signs and symptoms of such hydrocephalus are variable. They are often ascribed to non-specific delayed effects of the original lesion and thus not recognized. The diagnosis is established by pneumoencephalography.

5) The progressive hydrocephalus often produces an elevation of intracranial pressure but even in the presence of normal spinal fluid pressure, dramatic reversal of advanced neurological deficits and decrease in size of ventricles can follow cerebrospinal fluid shunt procedures.

6) The hydrocephalus secondary to the adhesive arachnoiditis evoked by the subarachnoid bleeding ceases to be reversible and becomes permanent after a period of time.

7) Treatment by ventriculomastoid shunt operations resulted in marked clinical improvement in 6 cases, impending fatality being averted in 3 of these.

8) The possible role of an adhesive arachnoiditis of the basal leptomeninges secondary to subarachnoid bleeding in the pathogenesis of infantile congenital hydrocephalus is discussed.

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SAFE ANGIOGRAPHY

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THE subject matter of this paper consists of cerebral angiography performed on 400 patients in the Department of Neurosurgery of the Royal Prince Alfred Hospital, to a total of 660 angiograms. All the procedures described were performed by the writer, using the same technique throughout.

Most of the patients were in the group aged 30–60 years, but 6 patients were less than 10 years old, and 8 patients were over 70 (the oldest being 85 years).

Angiography was employed as a diagnostic measure in patients suffering from many different provisional diagnoses, as seen in Table 1.

TABLE 1

Disorders for which angiography was used as diagnostic measure

Diagnosis	No. of Cases
Tumour suspect	144
Intracranial haematoma	21
Subarachnoid haemorrhage	74
Cerebral abscess	3
Cerebral thrombosis (suspected tumour)	11
Degenerative condition	50
Epilepsy (focal and otherwise)	41
Postoperative check angiogram	19
Miscellaneous	37
	400

METHOD OF ANGIOGRAPHY

For carotid angiography, the patient is placed with the head lying on a cassette box, and a sterile drape is placed over the chest and abdomen. The skin of the neck is prepared with solution of Metaphen, and a small amount of local anaesthetic is injected into the skin of the neck 1 inch above the clavicle, over the medial border of the sternomastoid muscle. The carotid artery to be cannulated is palpated, and a gauge 18 Luer-Lok needle is introduced so as to pass up the artery for $\frac{1}{2}$–1 inch at least. With practice this can be accomplished with ease, and blood will spurt from the needle. A flexible plastic tube, 5 inches long and $\frac{1}{8}$ inch in internal diameter, is used to connect the needle with a 20 cc. syringe, Luer-Lok fittings being used

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throughout to avoid the connections parting during the rapid injection of the dye. In the syringe are 15–20 cc. of a mixture of equal parts of sterile normal saline and 2.6 per cent sodium citrate solution. This solution is used to keep the needle free from clots, and produces, in the conscious patient, a rather unpleasant taste in the mouth (a valuable sign of correct positioning of the needle). Care is taken to fill all parts of the apparatus with fluid and so avoid injecting air bubbles. Blood flows back into the syringe and can be washed to and fro with the citrate solution, keeping the needle free from clots. Up to 120 cc. of this solution have been used with no untoward complications, in direct opposition to Sedzimir,[9] who gave 20 cc. as the maximum amount that could be given in this procedure with safety.

The head is postured and the roentgen-ray tube is aligned, and the syringe containing the dye is connected in place of the citrate solution. The injection is made rapidly and films are taken to show arterial and other phases. After anteroposterior and lateral views are demonstrated in turn by this method, further citrate solution is connected to the tubing and the needle is kept patent while the films are being developed, in case other views, such as oblique or cross-filling films, are required. When no further films are required, the needle is withdrawn and pressure is maintained over the artery for about 3 minutes. With this precaution, haematomata in the neck of sizeable proportions resulted in only 5 per cent of cases (30 patients), whilst moderate haematomata occurred in 8 per cent (48 cases) and small haematomata in 20 per cent (126 cases). In no case did respiratory embarrassment occur, and no haematoma persisted for more than 24 hours. Thus only temporary discomfort to the patient occurred.

For vertebral angiography a longer needle is used (such as for lumbar puncture), with a bevel of 45 degrees at its tip. The needle is introduced medial to the carotid bundle and is inserted between the transverse processes of the 4th and 5th or 5th and 6th cervical vertebrae at slightly less than a right angle to the neck. Injection technique is similar to that used in carotid angiography, except that the needle is withdrawn while the films are developed, as it is difficult to keep the needle in the lumen of the artery for more than 3 or 4 minutes without arterial spasm developing.

To prevent infection, all persons present wear gowns, caps and masks, the latter having a layer of cellophane between six layers of gauze. Gloves are not worn as they impair the sense of touch so necessary for successful needling of the artery. The hands are scrubbed for 5 minutes and dried. As will be seen later, infection was never encountered as a complication in this series.

Using the above techniques, good vascular filling and adequate roentgen-ray films were obtained in 95 per cent of cases. The main causes of failure in the other 5 per cent were faulty insertion of the needle in the artery, rapid development of a haematoma in the neck, movement of the head during exposure of the films, and occasional faults in the equipment.

CONTRAST AGENTS

The first 300 angiograms of the series were performed using Uriodone 35% (Diodrast). This was replaced by Diaginol 30% (sodium acetrizoate) for the remainder of the series. Both solutions gave good results but Diaginol produced less discomfort to the conscious patient, less stimulus to the anaesthetised patient and still good contrast in the films.

Each view was taken using an injection of 8 cc. only of the contrast agent. With this small volume, timing of the exposures required the close cooperation of the radiographer, but resulted in angiograms equal to and often better than those in which larger volumes of dye were employed. The absence of any serious complications was ascribed largely to the small amount of contrast agent of low concentration used.

As regards testing for sensitivity, this was discontinued when 100 cases had shown that sensitivity reactions were few and slight, and appeared unrelated to the results of skin or intravenous test doses.

RADIOGRAPHIC DETAILS

Most of the series of angiograms was taken using a Schonander machine with fixed tube-film distance of 25 inches (63.5 cm.). Cassettes measuring 12 inches (30.5 cm.) by 10 inches (25.4 cm.) with fine-grain screens were fitted into a cassette box on slides, the uppermost slide having a lead backing to protect the next cassette. The box was fitted with a Schonander stationary grid, and two or three cassettes could be used. In the majority of cases, one arterial and one venous film were exposed. For special indications, arterial, capillary and venous films were taken, such as in cases of arteriovenous anomalies, highly vascular tumours, and cases in which there was a very rapid circulation time.

Best results were obtained using a relatively long exposure (0.8–1.0 sec.) because of the small volume of contrast agent employed. The arterial film was exposed during the injection while 3 cc. of dye remained in the syringe and the venous film was taken 4 seconds later.

Exposures were made using 100 mA. and 54–62 kVP. (depending on the view taken of the skull). The tube used was oil-cooled, with a rotating anode. The operators were protected with lead-rubber removable screens about the patient's head, and wore lead-rubber full-length aprons.

About 30 angiograms of this series were performed in the general X-ray department of the hospital during overhaul of the Schonander unit. These were carried out using an ordinary tube-stand and other fittings, and their success illustrates the ease with which angiography can be performed with standard equipment used for general radiography.

ANAESTHETIC TECHNIQUE

Premedication of the patient is regarded as very important for successful angiography. The dosage recommended is shown in Table 2.

In addition, all patients for vertebral angiography were given papaverine gr. 2 (130 mg.) intramuscularly 45 minutes before the procedure, to abolish arterial spasm. A trial of this drug in carotid angiography produced no definite advantages in either the needling of the artery or in the films obtained, but both these factors were greatly enhanced in vertebral angiography. However, this drug was given in carotid angiography performed on patients with evidence of marked arteriosclerosis or with possible cerebral

TABLE 2

Premedication for angiography

Type of Patient	1½ hrs. before Procedure	¾ hr. before Procedure (Hypodermic Injection)
Average healthy adult	Pentobarbital sodium gr. 1½ (100 mg.)	Omnopon gr. ⅓ (20 mg.) Scopolamine gr. 1/150 (0.44 mg.)
Adult of slight build. Child over 12 yrs.	Pentobarbital sodium gr. 1½ (100 mg.)	Omnopon gr. ⅙ (10 mg.) Scopolamine gr. 1/300 (0.22 mg.)
Adult in fair health. Child aged 9–12 yrs.	—	Omnopon gr. ⅙ (10 mg.) Scopolamine gr. 1/300 (0.22 mg.)
Sick adult. Child under 9 yrs.	—	Atropine gr. 1/100 (0.6 mg.) or less according to age

thrombosis, and may therefore have been a factor in the prevention of lasting complications.

Both the premedication given and the choice of anaesthetic agents were dictated solely by the patient's state of consciousness and ability to cooperate. Table 3 shows the anaesthetics employed.

As vertebral angiography is a more painful procedure than carotid angiography, intravenous thiopentone anesthesia was always employed. For carotid angiography, however, local anaesthesia (2 per cent procaine) was used almost as often as general anaesthesia, and was found to be speedy and convenient to both patient and surgeon. Adequate premedication and careful explanation to the patient before injecting the dye rendered results with local anaesthesia very satisfactory.

Intravenous thiopental sodium was given in 206 cases, an average amount of 1.2 gm. being required for the procedure. No patient received more than 2.0 gm., and for heavily built adults, muscle relaxants such as Flaxedil (gallamine triethiodide) or Eulissin (decamethonium iodide) were used in combination with the thiopentone. A pharyngeal airway was used when necessary. Laryngeal intubation was found unnecessary, and therefore lighter anaesthesia could be used without producing coughing. There was also less apparatus about the patient's head. Oxygen given by an oro-

TABLE 3

Anaesthetics used in angiography

Type of Angiogram	No. of Angiograms	Local Anaesthesia	Thiopentone Sodium	Rectal Avertin	Avertin + Thiopentone	Ether
Carotid	630	272	306	28	21	3
Vertebral	30	—	30	—	—	—
	660	272	336	28	21	3

pharyngeal catheter was the only gas employed. Only 3 cases of laryngeal spasm occurred, all being relieved by blowing oxygen onto the vocal cords with a catheter. The fact that no deaths and no complications occurred in this large number of cases illustrates both the safety of the simple routine employed, and the competence of the Registrars in Anaesthesia who administered the anaesthetics.

Rectal Avertin (tribromomethanol), either alone or combined with intravenous thiopental sodium, was found rather unsatisfactory because of the variable response of different patients to a basal dose, a frequent fall in blood pressure, rendering needling of the carotid arteries difficult, and the long period of unconsciousness (up to 8 hours) following this type of anaesthesia.

For small children, ether was used in 3 cases with good results, but the risk of an explosion in the presence of electrical equipment must always be borne in mind.

Angiography under general anaesthesia was performed on patients with myocardial damage, severe hypertension, large cerebral tumours, on children of 8 years to adults of 70–80 years of age, all without complications. Nevertheless a laryngoscope and Magill's tubes and intermittent positive pressure respirator, stimulant drugs, antihistamine drugs, and apparatus to deal promptly with cardiac arrest were always at hand.

RESULTS OF ANGIOGRAPHY

Table 4 illustrates the diagnoses made possible by cerebral angiography.

In addition to these positive pathological angiograms, Table 5 illustrates cases in which a normal angiogram was of great value in the differential diagnosis and management of the case.

Angiography was found very useful in gauging progress and prognosis postoperatively, especially after surgical treatment of aneurysms, vascular tumours and subdural haematomata. With regard to angiography in cases of subarachnoid haemorrhage, this was usually performed as soon as possible, and in many cases it was done within the first 24 hours of the illness. No patient died because of angiography and the information supplied by the procedure proved so valuable in the management of the case that immediate angiography is a well accepted routine in this department. The results obtained from early angiography with the technique described above show that of 74 cases of subarachnoid haemorrhage, aneurysms were revealed in 44, or 60 per cent. Of the other 40 per cent, repeated angiography, 1 week or more later, still revealed no aneurysm, even on the basi-vertebral system. These figures present a contrast to those of Magladery[5] who found 66 per cent of negative angiograms in the first week and 50 per cent after that, and who stated that death followed soon after angiography in 10 cases.

With cerebral tumours, angiography very often enabled a diagnosis of the exact type of neoplasm, such as glioblastoma multiforme, because of the typical vascular "pattern" produced. The value of angiography in lesions

TABLE 4

Diagnoses made by cerebral angiography

Condition	No. of Cases	Types
Intracranial tumour	80	31 Frontal 22 Temporal 16 Parietal 1 Occipital 3 Multiple secondary 2 Mid-brain & cerebellar 5 Pituitary
Intracranial haematoma	21	1 Extradural 13 Subdural 7 Intracerebral
Arterial aneurysm	44	15 Internal carotid 17 Anterior communicating 8 Middle cerebral 2 Anterior cerebral 1 Basilar 1 Multiple
Arteriovenous malformation	2	
Cerebral abscess	3	
Cerebral thrombosis	11	2 Internal carotid 2 Anterior cerebral 7 Middle cerebral
Total	161	

about the pituitary fossa has been demonstrated by Money and Vanderfield,[6] and their findings were illustrated again in the 5 cases of pituitary tumour and in several cases of aneurysms about this region in the present series.

Carotid angiography in cases of subtentorial tumours was of use both in eliminating a supratentorial lesion and in demonstrating a hydrocephalus as shown by an exaggerated sweep of the anterior cerebral arteries around the dilated anterior horn of the ventricle. Vertebral angiography gave rather disappointing results as far as showing a pathological tumour circulation and was used mainly in cases of subarachnoid haemorrhage.

TABLE 5

Cases in which normal angiograms aided in diagnosis

Condition	No. of Cases
Degenerative condition	50
Subarachnoid haemorrhage	30
Epilepsy	41
Tumour suspect	64

COMPLICATIONS OF ANGIOGRAPHY

In this series there were no deaths, no permanent sequelae, nor were any transient complications encountered, except for the occurrence of cutaneous rashes in 7 cases (1 per cent), relieved immediately by antihistamine drugs. The frequency of complications in other series is shown in Table 6.

The absence of complications was ascribable in part to the small volume of contrast agent used, but also to the fact that any patient who was shown to have a large space-occupying lesion was treated surgically as soon as possible, usually within 12 hours. In this way any possible cerebral oedema caused by the angiography was not allowed to increase an already raised intracranial pressure for more than a short time.

TABLE 6

Comparison of frequency of complications in various series

Date	Authors	No. of Angiograms	Complications		
			Transient	Permanent	Deaths
1950	Bull[2]	1,000	?	?	3
1951	Dunsmore *et al.*[4]	147	7	4	3
1951	Curtis[3]	720	6	0	1
1952	Abbott *et al.*[1]	174	8	4	5
1953	Rowbotham *et al.*[8]	430	18	12	3
1954	Perese *et al.*[7]	234	19	3	7
1954	Ver Brugghen[10]	150	0	0	0
1955	Sedzimir[9]	273	0	0	0
1956	Present series	660	0	0	0

SUMMARY

1. A safe method of carotid and vertebral angiography is described. It was performed 660 times on 400 patients without any complications or mortality.

2. The choice of anaesthesia is discussed, and the dosages of premedicating and anaesthetic agents are presented.

3. The results obtained are listed and the value of the procedure in various conditions is discussed.

4. Complications in other series are compared with the present series, and their absence in this series is attributed to the small volume of contrast agent used, the careful premedication and choice of anaesthetic, and to the repeated use of the technique of percutaneous angiography described above.

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INFARCTIONS SIMULATING BRAIN TUMOURS IN THE POSTERIOR FOSSA

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IT HAS long been recognized that occlusions of the internal carotid artery and its branches may cause circulatory disturbances in the involved area of the brain, resulting not only in malacia and atrophy, but also temporarily producing oedema. In the first days after the accident this may cause increased intracranial pressure.

In these cases a diagnosis of intracranial haematoma or other type of expanding lesion has been made not infrequently, and the patients have sometimes been operated upon, usually without preoperative arteriograms.

In the posterior fossa, expanding lesions often have to be treated acutely. A pneumoencephalogram or a ventriculogram may have established the expanding nature of the lesion. Vertebral angiography is more rarely performed. Recently we had 2 patients demonstrating the difficulties in diagnosing the true nature of the expanding lesion; 1 of these was, however, suspected of having a vascular occlusion before the operation was performed.

CASE REPORTS

Case 1 (050716/55 S.S.). A 50-year-old man, previously healthy, first complained of pain and numbness of his left arm in the middle of October 1955. He improved following roentgen-ray treatment. On Nov. 18, 1955 he began to have occasional dizziness, nausea and vomiting, and the following day there was onset of headache in the right parieto-occipital region.

He was examined in another hospital on Nov. 21, 1955. Blood pressure was 140/60. There was slow nystagmus on gazing to the left. Sensibility was normal. Babinski sign was positive—? bilaterally. Lumbar puncture revealed normal spinal fluid under normal pressure. That night the patient became disoriented and restless. The next morning he was drowsy, and did not answer but reacted to painful stimuli. He was admitted to our hospital on Nov. 22, 1955.

Examination. Blood pressure was 170/95. He was sometimes able to answer "yes" and "no" and to move all extremities spontaneously with full power. There was extreme conjugate deviation of the eyes to the right. Pupils were normal, the right pupil sometimes being slightly larger. Fundi were normal. Abdominal reflexes were absent bilaterally. Babinski sign was positive on the right, and absent on the left. Roentgenogram of the skull and left carotid angiogram were normal (no filling of the posterior cerebral artery).

Course. His condition deteriorated, he reacted hardly to painful stimuli, and his breathing became shallow and irregular. Neurosurgical consultant suspected oc-

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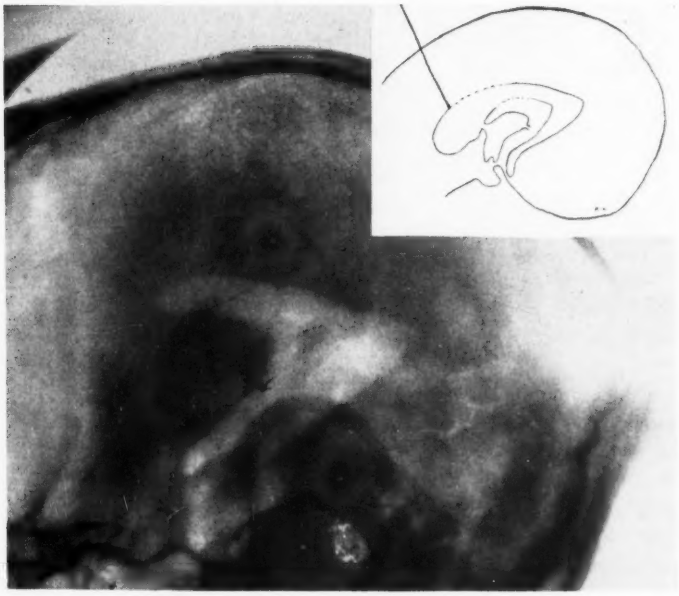

FIG. 1. *Case 1.* Ventriculogram. The Sylvian aqueduct is filled only to the extent of a few mm. from the 3rd ventricle.

clusion of the basilar artery, and after a cisternal puncture (no cerebrospinal fluid obtained) an unsuccessful attempt was made to perform vertebral angiography. Then ventriculography showed normal lateral ventricles, but the Sylvian aqueduct was filled only to the extent of a few mm. from the 3rd ventricle (Fig. 1).

Operation. On Nov. 22, 1955, under nitrous oxide-oxygen anesthesia, exploration of the posterior fossa was performed with removal of the arch of the atlas. The dura mater was very tense in spite of a ventricular puncture. When the dura mater was opened the gyri of the left cerebellar hemisphere appeared flattened and broadened, and the surface of the medial inferior area "exploded" with emptying of a yellow-white porridge-like substance (Fig. 2). The malacic cavity was about the size of a plum. Biopsy showed only malacia.

Course. Half an hour after the operation, when the patient was turned in his bed, he suddenly expired.

Autopsy. Pulmonary emboli from a femoral venous thrombosis were revealed as the cause of death. Four small ulcerations in the ventricular mucosa were found. There were remnants of the left cerebellar hemisphere in the medial, superior and anterior regions, surrounding the operative cavity as a shell. Part of the left tonsil was also destroyed and there was a pressure cone on the right side. There were no signs of tumour; the medulla and pons were normal. The posterior inferior cerebellar artery and the vertebral artery on the left side were thrombosed (Fig. 2). The anterior inferior cerebellar artery was a little smaller on the left than on the right side.

Microscopical examination revealed oedema in the "lateral medullary area" below the restiform body.

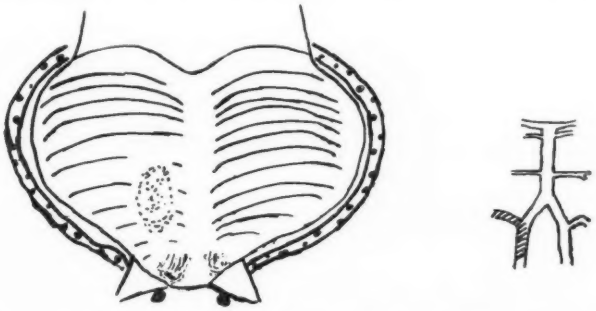

FIG. 2. *Case 1.* (*Left*) Operative sketch, showing flattened and broadened gyri. The location of the rupture of the malacic cavity in the left hemisphere is marked. (*Right*) Postmortem sketch indicating the occlusion of the left vertebral and posterior inferior cerebellar artery.

Case 2 (100128/55 S.S.). A 45-year-old man, previously healthy, had complained of slight headaches now and then since the middle of November 1955. On Dec. 5, 1955, he began to have severe headache, which developed during a few hours. The headache was most severe in the occipital region but was also in the temporal and frontal regions. He felt a little dizzy but had no other complaints.

On Dec. 6, 1955, he was examined in another hospital. Blood pressure was 200/100. There was slight cervical rigidity but there were no other neurological abnormalities. Eyegrounds were normal. Blood sedimentation rate was 10 mm./h. Lumbar puncture revealed colourless cerebrospinal fluid under 22 cm. pressure; protein was 70 mg. per cent; Wassermann reaction was negative. He had no fever, but the leucocyte count in the blood was slightly increased. On Dec. 8, 1955 he became drowsy with nuchal rigidity. Blood pressure was 155/110. Lumbar puncture yielded

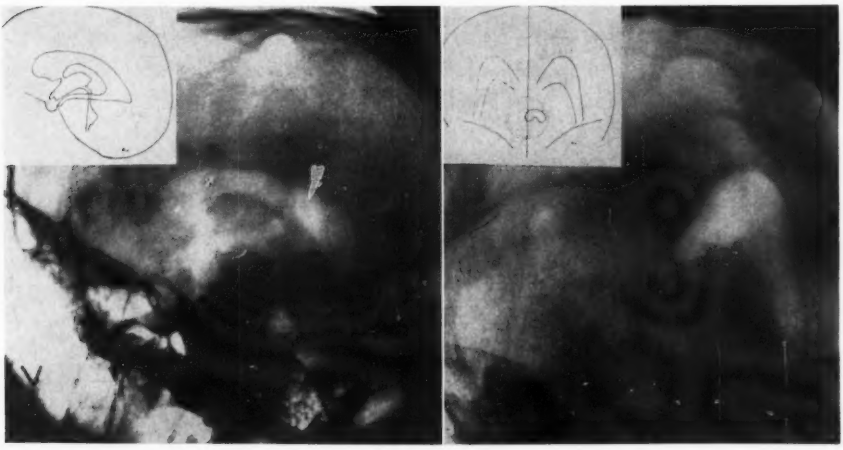

FIG. 3. *Case 2.* Ventriculogram showing sharply bent Sylvian aqueduct and dislocation of the 4th ventricle to the left. (At operation the appearance of the right-sided softening was similar to that shown in F.g. 2.)

colourless fluid under 26 cm. pressure and protein was 46 mg. per cent. The following day he was slightly disoriented, and sleeping but arousable. Babinski sign was not present. He was admitted to our hospital on Dec. 9, 1955.

Examination. Blood pressure was 160/100. Pulse rate was 72. The patient was drowsy and the gag reflexes were weak. The right optic fundus was normal, and the left showed oedema of the nasal half of the disc.

Course. The next day he was temporarily stuporous. Blood pressure was 170 /100. There was paralysis of upward gaze and a horizontal nystagmus on gazing to the right. Electroencephalogram revealed diffuse abnormality, most marked in the left temporal region. Roentgenogram of the skull was normal. Encephalography by the lumbar route was attempted (after precautious trepanation); only the basal cisterns became filled; the cisterna pontis was very narrow. Because of a possible slight improvement he was observed until Dec. 14, 1955, when ventriculography was performed. The aqueduct was bent sharply and the 4th ventricle was dislocated to the left (Fig. 3).

FIG. 4. *Case 2.* There is irregular narrowing of the proximal part of the right vertebral artery. Angiography was done by means of a brachial catheter into the subclavian artery. (Arrows indicate catheter.)

Operation, Dec. 14, 1955. After ventricular puncture the dura mater was opened. The right cerebellar hemisphere was slightly increased in volume and its surface was markedly yellow. Under the cortex of the medial inferior part of the convexity a walnut-sized cavity was found, filled with a creamy, yellow-white substance; there were also some harder pieces. Biopsy report: malacia.

Postoperative course was uneventful. His gait was staggering, with possibly slight deviation to the left. There was slight papilloedema bilaterally. Upward gaze became much improved.

An attempt to perform right-sided vertebral angiography was made by means of a brachial catheter, but it did not slip into the vertebral artery; injection in the subclavian artery with brachial compression revealed an irregular stenosis of the first part of the vertebral artery (Fig. 4). There was good filling of the first part of the artery distal to the stenosis, but no satisfactory picture was obtained of the intracranial branches.

One month after the operation the patient had no subjective complaints. Gait was normal. Funduscopic examination revealed no papilloedema on the right, slight on the left side. There was slight right-sided paresis of the soft palate and pharynx; the gag reflex was absent on the right side.

DISCUSSION

The "classical" clinical picture of an occlusion of the posterior inferior cerebellar artery has been described as the syndrome of Wallenberg. The underlying pathology is a softening of the lateral part of the medulla oblongata (mostly on the left side), but involvement of the cerebellar hemispheres is usually not reported. The last phenomenon has been ascribed to arterial anastomoses on the cerebellar surface, particularly between the anterior inferior cerebellar artery and the posterior inferior cerebellar artery. In the 2 cases presented here, there were some symptoms and signs pointing to a lesion of the brain-stem area, but not very characteristically or markedly and we were most impressed by the symptoms caused by the lesion of the cerebellar hemispheres.

Softening of the inferior part of the cerebellum has usually been explained as the result of occlusion of the lateral branches of the posterior inferior cerebellar artery (e.g., Haike and Lewy[3]), but in our Case 1 the main stem of the posterior inferior cerebellar artery and the adjacent part of the vertebral artery were occluded. The syndrome of Wallenberg varies in its details, and, moreover (see Schwarz[9]), the whole lateral part of the medulla may be supplied by the anterior spinal artery or the vertebral artery. Schwarz[9] reported a case in which a cerebellar lesion was associated with the medullary lesion, and Case 10 of Olivarius[7] was similar, but vascular changes probably present were not reported. This was also the situation in a patient reported by Michael[6] (in a footnote) with only a cerebellar softening at autopsy. However, Schaffer[8] reported a patient (Case 4) with an obliterated posterior inferior cerebellar artery, in whom only the cerebellar hemisphere was anatomically involved, and Thomas[10] found thrombosis of this artery in a case in which there was "absence of microscopical lesions—no softening of the medulla." In our Case 2 only a very narrow proximal part of

the ipsilateral vertebral artery was visualized on the arteriogram; the area of softening was within the distribution of the posterior inferior cerebellar artery.

From a clinical point of view, however, the course of the disease was even more important than the precise localization of the symptoms in these cases. The immediate prognosis of the Wallenberg syndrome is usually good and in not a few cases full restitution occurs. Consequently there is reason to ascribe an impairment in the condition of such a patient to another cause than the original local vascular disturbance. In a review of the clinical course in 28 patients, Lewis *et al.*[4] wrote: " . . . in two cases, death was considered due to the PICA thrombosis itself or to an undiagnosed fulminant complication within 48 hours of the onset of the original lesion." [No autopsy.] Goodhart and Davison[2] described a patient (Case 4, with also an older middle cerebral artery lesion) who died after having presented a course similar to that of our Case 1, but with less extensive destruction of cerebellar tissue.

The increased intracranial pressure in these cases might be caused by the compression and dislocation of the aqueduct and the 4th ventricle, but also by the increased volume of the involved area itself. Some authors (see Atkinson[1]) also assume that an increased intracranial pressure (from "cerebral swelling") and increased blood pressure are caused by a lesion in the tegmental area of the pons, but this has been discussed mostly in connection with occlusions of the anterior inferior cerebellar artery; moreover, the "vasomotor centers" in the mesencephalic and medullary areas are fairly complex and have different functions (Lindgren[5]). Subsequently the effect of a lesion in this territory is difficult to predict.

The radiological examination necessary to indicate the expanding nature of the lesion is pneumoencephalography; vertebral arteriography is not without danger in these cases and is more of academic interest. In the writer's opinion, surgical intervention is justifiable in some cases of cerebellar lesions following occlusions of the main arteries in the posterior fossa, i.e., when they start to cause increased intracranial pressure.

SUMMARY

Vascular occlusions of the cerebral arteries may cause tumour symptoms with increased intracranial pressure in the acute stage. This is rather well known with respect to occlusions of the internal carotid artery.

Two cases presenting a clinical and roentgenological picture of a tumour in the posterior fossa are reported. At operation softening in a cerebellar hemisphere with increased intracranial pressure was found. Autopsy in one case revealed a thrombosis of the corresponding vertebral and posterior inferior cerebellar artery. The other patient showed an irregular narrowing of the vertebral artery at a postoperative angiography.

The implications of the findings are discussed, particularly the variation of the vascular supply in this area. Surgical intervention is indicated when infarction in the posterior fossa gives rise to increased intracranial pressure.

ADDENDUM

Since the preparation of this paper, B. Fairburn and L. C. Oliver (*Brit. med. J.*, 1956, *1:* 1335–1336) have described 3 patients (men) with cerebellar softening, one of whom was operated upon in the acute stage, and resembled our Case 2.

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EPILEPTIC EFFECTS OF INTRACISTERNAL ALUMINA CREAM IN MONKEYS*

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APPLICATION of alumina cream by disc or injection into a cerebral hemisphere is an effective method for the production of chronic experimental epilepsy in monkeys.[7] The present study was undertaken to determine the comparative effectiveness of alumina cream injected into the cerebrospinal fluid of the cisterna magna.

METHOD

Alumina cream prepared in the laboratory was centrifuged and the sediment was injected into the cisterna magna of 5 young healthy *Macaca mulatta* (3.4–4.4 kg.). Approximately 0.2 to 0.5 ml. of cisternal cerebrospinal fluid was withdrawn through a 21 gauge hypodermic needle, mixed with 0.2 to 0.3 ml. of alumina cream sediment and slowly reinjected. The injection procedure was performed on animals under Nembutal anesthesia, seated with head and neck flexed. Immediately following the intracisternal injections, the monkeys were placed prone on a flat surface and allowed to recover from the effects of anesthesia without further positioning or restraints. In 1 animal (#811) a second intracisternal injection of alumina cream sediment was made 9 months after the first injection. Intracerebral multiple injections of saline had been made 20 months earlier in 1 monkey (#761). In another monkey (#701) ligation of the right middle cerebral artery had been made (2 years before), followed by contralateral multiple intracerebral Aquaphor injections (1 year before) without significant clinical residuals at the time of intracisternal injection. Clinical observations and electroencephalographic studies were made on all animals. Activation of clinical convulsions by injection of intramuscular pentamethylenetetrazole (Metrazol) was sometimes employed.[1] The brains and spinal cords were removed for gross and histologic examination in 4 of the monkeys; the remaining animal (#812) still survives (12+ months following intracisternal injection).

RESULTS

Epileptic seizures occurred in 4 of the 5 monkeys 15 to 27 days after intracisternal injection of alumina cream. Two of the animals (#806, #761) died in status epilepticus on the 2nd and 6th days respectively following onset of their clinical seizures (Table 1). Two others (#812, #701) exhibited spontaneous epileptic seizures or could be readily provoked to seizure by prodding or intramuscular Metrazol (8–24 mg./kg.) for 9 and 10 months

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TABLE 1

Effect of intracisternal alumina cream in Macaca mulatta

Monkey No.	Previous Treatment	Intracisternal Alumina Cream, ml.	Clinical Features	EEG Changes
806	—	.2	Convulsions 23 days later with status epilepticus and death in 48 hrs.	Status. Spike and wave pattern
812	—	.2	Convulsions 25 days later. Clinical activation by photic stimulation. Spontaneous seizures. Survived 12 mos. to date	Diffuse spiking; increased slow background
811	— Intracisternal alumina 9 mos. prior	.2 .2	No change Onset 16 days later: weakness, alteration of consciousness and body jerks. Sacrificed 4 days later	Slight slowing Slowing on 15th day
761	Multiple intracerebral saline inj. 21 mos. prior	.25	Convulsions 15 days later. Died in status on 20th day	Status
701	Multiple intracerebral Aquaphor (Lt.) inj. and middle cerebral ligation (Rt.) 12 mos. prior	.3	Convulsive response to i.m. Metrazol (16 mg./kg.) 29 days later and thereafter. Survived 9 mos.	Mild slowing

respectively. One animal (#811) showed no spontaneous clinical seizures or epileptic response to intramuscular Metrazol (32 mg./kg.) for 9 months. Sixteen days following a second intracisternal injection of alumina cream sediment an abrupt change in clinical status occurred, characterized by debility, staggering gait, intermittent jerks of body and extremities, and impaired level of consciousness up to time of sacrifice 4 days later.

Convulsive seizures were usually tonic-clonic generalized motor in type, although focal motor seizures with Jacksonian spread and myoclonic jerks were also noted. In one monkey (#812) seizures featured by salivation, twitching of face, turning of head to the side, chewing mouth movements and irregular jerkings of the extremities occurred during the first month following onset of epileptic seizures. When seizures were frequent or the animal appeared to be in status epilepticus, monkeys appeared dazed, incoordinate and weak between clinical episodes.

The electroencephalograms in 2 monkeys (#806, #761) that died in status epilepticus, showed diffuse rapid spike, and spike and slow wave convulsive discharges of varying intensity often followed by isoelectric periods (Fig. 1). The principal changes noted in the resting patterns of the other animals was

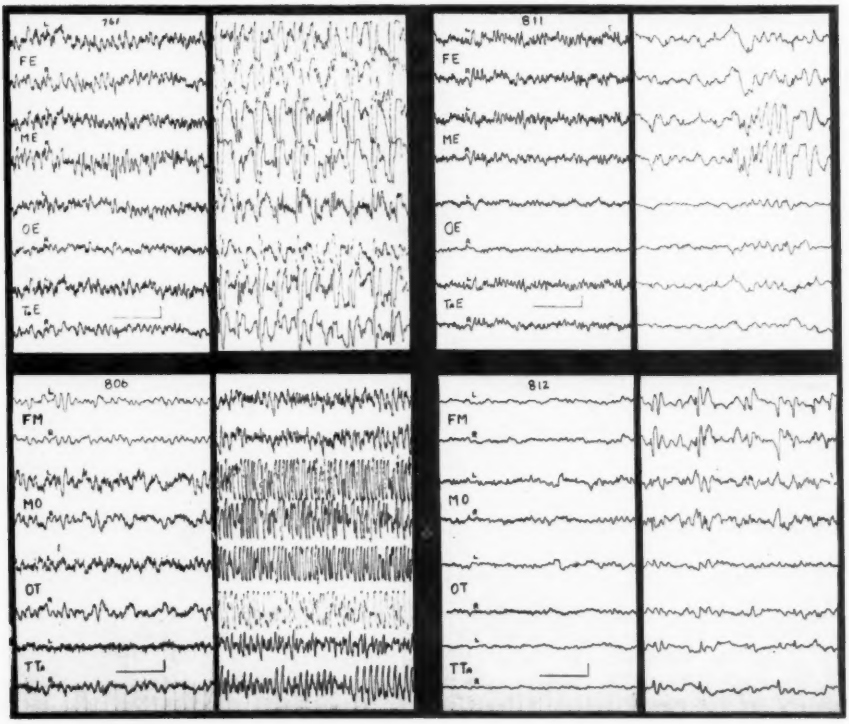

FIG. 1. Electroencephalograms before (left) and after (right) intracisternal injection of alumina cream in 4 monkeys. #761: 15 days after; #806: 23 days after; #811: 15 days after 2nd injection; #812: 5¾ months after. F = frontal; M = motor; O = occipital; T = temporal; Ta = anterior temporal; E = ipsilateral ear; L = left; R = right. Calibration: 1 second (horizontal); 50 microvolts (vertical).

an increased amount of theta waves and random spike and sharp wave formations. In one monkey (#812) clinical convulsive seizures were precipitated by photic stimulation on a few occasions.

No significant ventricular dilatation, hydrocephalus or myelomalacia was found on examination of the brains and spinal cords of 4 monkeys. Histologic examination in the case of a monkey that died in status epilepticus 23 days after injection (#806) disclosed a low-grade chronic diffuse leptomeningitis, which was most intense in the meninges of midbrain and brain stem. Demyelinization of ventral portions of cerebral hemispheres and midbrain was apparent in coronal sections stained by the Weil method. Alumina cream within macrophages was noted in the subarachnoid spaces and meninges of the brain, spinal cord and particularly at the base of the brain stem; some was also noted about spinal nerve roots and the cauda equina.

DISCUSSION

Cisternal injection of alumina cream induced epileptic seizures in 4 of 5 monkeys after a latent period of 15 to 27 days. The epilepsy produced in

these animals resembled that seen after the direct application of alumina cream to the cerebrum by disc or injection: in both groups there was usually a latent period of weeks, a tendency for seizures to be most frequent soon after their onset with a likelihood of status epilepticus to occur early, susceptibility to activation of clinical seizures by prodding or Metrazol, and similar patterns of clinical seizures, electroencephalographic changes and behavioral alterations.

In 1 monkey (#812) the occasional effectiveness of photic stimulation (15 flashes per sec. frequency) in provoking clinical seizures was also noted.

Hurst's studies[3,4] made on a group of more than 40 monkeys demonstrated that a variety of dissimilar irritants introduced intracisternally were capable of producing damage to superficial nervous structures, cellular proliferation and fibrosis in the pia arachnoid, and necrosis of media and often of adventitia of meningeal arteries. Convulsive seizures occurred in 2 monkeys of his series. In 1 of 2 monkeys injected with a non-ionic detergent (alkylated phenol-ethylene-oxide condensate) epilepsy developed 17 days post-injection with death 2 days later and in 1 of 3 monkeys injected intracisternally with an antiseptic (1:6 Di-4′-chlorophenyldiguanidohexane) epileptic seizures and death occured in 24 hours after injection. According to McLaurin and co-workers,[8] intracisternal injections of kaolin (aluminum silicate) in dogs can produce severe chronic adhesive arachnoiditis with resultant communicating hydrocephalus and myelomalacia of the cervicothoracic spinal cord. Convulsive seizures, however, were apparently not observed in these animals nor were they mentioned in the earlier report of Dixon and Heller,[2] who used intracisternal injections of kaolin in dogs to produce elevated intracranial pressure. The meningeal reaction in our case (#806) was much milder than that described as following intracisternal kaolin and/or Pantopaque in dogs. The possibility that this difference in meningeal reaction was influenced by the species used, the type and concentration of irritant chemical and the time interval after injection must be considered. Further studies of the pathological alterations induced by intracisternal alumina cream in monkeys are in progress.

The use of intracisternal injections for the production of chronic epilepsy in monkeys has not been previously described. Although the present technique has dealt principally with the effects of single intracisternal injections of alumina cream, we feel that multiple, repeated intracisternal injections, or the injection of other quantities or concentrations of alumina cream or related compounds, may also prove effective in producing epilepsy.

The capacity of alumina cream to exert an epileptogenic effect when introduced into the cerebrospinal fluid of the subarachnoid spaces by intracisternal injection or when applied by disc to the cerebral surface indicates that an epileptic response may result without application of the alumina cream directly into the nervous tissue parenchyma. Our results in this respect resemble those obtained by Kennard,[5,6] who showed that alumina cream could exert an irritant effect when injected into the spinal subarachnoid spaces of the cat. These findings also suggest that it may be necessary

to consider dispersion or reflux into cerebrospinal fluid from cerebral sites of application as a factor affecting the epileptic reaction of monkeys treated with alumina cream.

SUMMARY

The clinical and electroencephalographic changes associated with injections of alumina cream into the cisterna magna of 5 monkeys are described. Epileptic seizures occurred in 4 which resembled in many features those of chronic experimental epilepsy produced by application of alumina cream directly to a cerebral hemisphere.

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THE USE OF PHENOLSULPHONPHTHALEIN IN THE CLINICAL EVALUATION OF HYDROCEPHALUS*

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THE use of phenolsulphonphthalein to study the cerebrospinal fluid system was introduced by Dandy and Blackfan in 1913.[4,5] They assumed this dye labeled cerebrospinal fluid as a whole and that it could be used as a measure of the absorption rate of cerebrospinal fluid as well as communication between the ventricles and the spinal subarachnoid space. However, new information about the cerebrospinal fluid which has been obtained through the use of radio-active and stable isotope tracers[1–3] has made it necessary to re-examine the basic principles involved in this test to determine whether or not the interpretations usually placed on it are valid. This paper is a study of this test based upon some 301 dye studies done on various patients of the neurosurgical service of The Children's Medical Center, Boston.

MATERIALS AND METHODS

The dye studies considered here have been carried out on neurosurgical patients (mostly under 2 years of age) as a part of their clinical investigation. The test has been done in very nearly the manner described originally, except that absorption of dye was studied only after ventricular injection.

Bilateral ventricular taps were done after investigation of the subdural spaces; simultaneously a lumbar puncture was done. The cerebrospinal fluid pressure of both the lumbar and ventricular regions was measured and samples of fluid were taken for study. Then 1 cc. of a neutral solution containing 6 mg. of phenolsulphonphthalein was placed into one lateral cerebral ventricle. The cerebrospinal fluid was allowed to drip from the lumbar needle for 30 to 60 minutes onto a gauze which had been soaked in ammonia. The time elapsed between injection of the dye and its appearance in the lumbar fluid was taken as the "communication time." Dye was usually allowed to drip for some short period after this to see if the dye became more concentrated. In some cases in which no dye appeared in the 30- to 60-minute period repeated lumbar punctures were carried out at intervals of 1 or more hours for a 24-hour period. After all of the needles were removed, the urine was collected for a period of 24 hours with specimens being taken separately at intervals of 2, 3, 6, 8, 12 and 24 hours. The number of specimens from any

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one patient depended upon the urinary output, which was enhanced by forcing fluids orally and occasionally giving them parenterally. The amount of phenolsulphonphthalein excreted during each interval was calculated as a percentage of the total amount given. In a small group of patients the disappearance of the dye from the cerebral ventricles was followed as well as the excretion of the dye in the urine. This technique allows a measure of the volume of cerebrospinal fluid by measuring the dilution of the dye.

The time required for half of a tracer (in this case, phenolsulphonphthalein) to be removed can be used as a measure of its turnover or exchange rate. This time, known as the "half time," will be used in the discussion of the data being considered here.

RESULTS

Communication of the Ventricles with the Subarachnoid Space. In 130 of the 301 patients tested, communication of the ventricles with the lumbar subarachnoid space was demonstrated by dye. The time of communication was recorded in 123 cases, and in 7 communication was found but the time was not recorded. These times were fairly well grouped and have been examined by standard statistical methods. The range of communication time was 0.3 minute to about 24 hours. The communication times over 1 hour are not strictly comparable as they were determined by multiple puncture and not steady flow. The mean time for dye to appear in the lumbar subarachnoid space after injection into the cerebral ventricles was 6.7 minutes with a 0.3 standard deviation from the mean of 4–6 minutes. This suggests that in any child who shows no communication within 20 minutes (mean plus 3 standard deviation) there is probably inadequate communication or the flow of cerebrospinal fluid is obstructed, and any patient who does not show signs of communication within 15 minutes should be regarded with great suspicion. It should be pointed out here that very often when doing this test the spinal fluid will stop flowing after a few minutes even though the needle is still in the subarachnoid space. This has been found to be a true indication of noncommunication between the ventricles and lumbar subarachnoid space.

There were 14 patients in this group in whom communication of the ventricles with the spinal subarachnoid space could not be confirmed by air ventriculography or who were thought to have obstructive hydrocephalus on other clinical grounds. In 5 of these patients, dye was not found in the lumbar cerebrospinal fluid for several hours, and this would be considered as evidence of noncommunication by the time limits given here. Three of these patients had obstruction of the foramina of Magendie and Luschka, 1 had an aqueductal atresia proven at operation, and in 1 no surgery was carried out. Four of the other 9 patients with relatively shorter communication times were found to have obstruction of the foramina of Magendie and Luschka which was seen in the air studies. The passage of dye in these patients was slow (over 10 minutes in all but 1) and the dye never reached a

high concentration in the lumbar fluid. Two of the remaining 5 were not explored, so nothing certain is known about the state of the aqueduct or the posterior fossa. In the remaining 3, exploration was carried out. Only 1 of these was found to have an occluded aqueduct through which neither fluid or dye would flow nor could a catheter be passed. In 1 there was considered to be a partial aqueductal stenosis at operation, as fluid could be seen to be flowing through it easily but a catheter would not pass. In 1, the aqueduct was found to be open at operation and no other lesion was described.

Comment. From the review of these tests it appears that communication is most quickly established if the fluid is allowed to drip continually from the lumbar needle. This should be allowed to continue for at least 15 minutes or until no more fluid will flow. If dye has not appeared in this time, or if the subarachnoid space runs dry, it is fairly certain that there is inadequate communication between the ventricles and the lumbar region. If the air studies do not confirm the inadequacy of communication found by the dye test and do not show some other lesion, both the air studies and the dye test should be carefully reviewed and perhaps repeated before deciding upon the diagnosis and planning therapy.

Removal of Phenolsulphonphthalein from the Cerebrospinal Fluid. The absorption and urinary excretion of phenolsulphonphthalein were adequately observed in 159 patients. Of these, 85 had communicating hydrocephalus, 71 had noncommunicating hydrocephalus, and 3 had cerebrospinal fluid systems of relatively normal size without increased pressure. Dye appeared in the urine within 2 hours in all types of patients, but not in equal concentration. No attempt was made to determine how fast it appeared in the urine. The normal children eliminated almost all of the dye within 24 hours. The hydrocephalic patients were much slower as a group but there was great variation among patients. Those with communicating hydrocephalus had a mean 24-hour dye excretion of 33 per cent with a range from 6 per cent to 86 per cent. Those with noncommunicating hydrocephalus had a mean 24-hour dye excretion of 23 per cent with a range of 9 per cent to 53 per cent. While these mean figures are in agreement with the original data of Blackfan and Dandy, the range of variation among the patients of each group is so great and there is so much overlap that the test is rendered valueless in differential diagnosis for the individual patient. These data are graphically summarized in Fig. 1.

These results do suggest that absorption of phenolsulphonphthalein is slower in hydrocephalus, but a careful examination of the concepts upon which the test is based will show that this conclusion is not true.

This test is based upon the assumption that phenolsulphonphthalein labels cerebrospinal fluid as a whole. However, recent tracer studies[1–3] have shown that most of the constituents of cerebrospinal fluid move at their own individual rates and no one substance that has been studied can be used as a measure of cerebrospinal fluid *in toto*. Since there is no evidence to show that phenolsulphonphthalein is a special case and does in fact represent

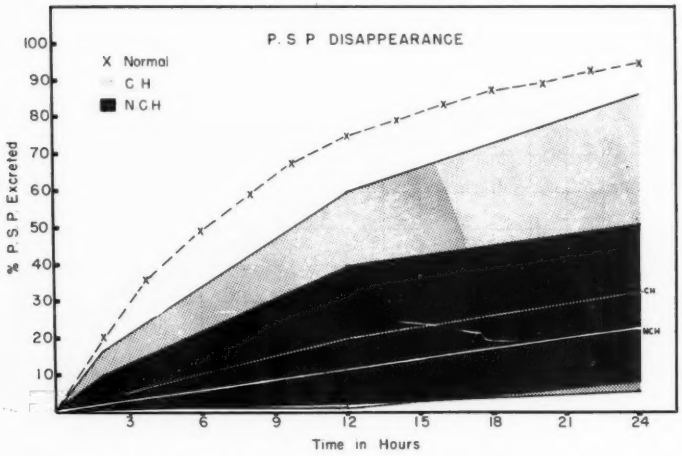

FIG. 1. The removal of phenolsulphonphthalein from the cerebrospinal fluid after injection into the cerebral ventricles of normal and hydrocephalic infants. The cross-hatched area, which represents the spread of results obtained in patients with communicating hydrocephalus, completely covers the solid area, which represents the spread of patients with noncommunicating hydrocephalus. This chart is based on data obtained by studying the time of appearance of the dye in the urine.

cerebrospinal fluid as a whole, this idea must be abandoned in favor of the concept that what is being measured is the rate of the handling of phenolsulphonphthalein alone. This does not mean that phenolsulphonphthalein cannot be an indication of the state of the general absorptive mechanism of the cerebrospinal fluid, but before it is so used it must be demonstrated that the absorption of phenolsulphonphthalein is, in fact, altered in hydrocephalus.

As the test has been performed, the same quantity of dye has been used regardless of the size of the ventricle. This results in wide variation of the concentration of the dye in the cerebrospinal fluid, a fact that would markedly affect the rate of its appearance in the urine even if there were no difficulty with absorption of the cerebrospinal fluid. The 6 mg. of dye put into a normal ventricular system of 10-cc. capacity would result in a concentration of 60 mg. per cent, while the same amount in a hydrocephalic patient with 1000 cc. of cerebrospinal fluid would result in a concentration of only 0.6 mg. per cent. This difference in concentration would cause the appearance of phenolsulphonphthalein in the urine to be very slow even if the rate of cerebrospinal fluid absorption were the same in both patients.

This can be clearly demonstrated by a simple analogy. Suppose there is in a bathroom a wash basin of 5 liters' capacity and a bathtub of 500 liters' capacity. Suppose further that both of them are full but that water is continually running into both of them and out at the same rate of overflow, say 2.5 liters per minute (Fig. 2). Now suppose the same amount of some dye is introduced into the bathtub and the wash basin. The concentration of

the dye in the wash basin will be 100 times that in the tub, just as the concentration of the dye is much greater in the normal ventricle than in the hydrocephalic ventricle. Now if the outflow is collected, it will be seen that in 1 minute the wash basin will have had half its volume replaced and therefore about half the dye will have come out: so it can be said that the dye "half time" of the basin is about 1 minute. The bathtub, on the other hand, will require 100 minutes for half its volume to be run in and an equal amount out, so that here the dye "half time" will be 100 minutes. Using removal of the dye alone without considering concentration as in the phenolsulphonphthalein test, one might say that flow in the bathtub was only 1/100 as fast as that in the wash basin, although in fact the rates of flow are equal. If the percentage of the dye removed were plotted against time for both basin and tub, the curves would appear as shown in Fig. 2, which are indeed similar to the differences seen between the phenolsulphonphthalein tests of the normal and the hydrocephalic patients (Fig. 1). It is apparent from this that in the phenolsulphonphthalein test as it is done, neglecting the concentration of the dye in the cerebrospinal fluid, a marked slowing of the appearance of the dye in the urine is to be expected in hydrocephalic states, whether or not the absolute absorption of the dye has been altered.

The effect of hydrocephalus on the absolute rate of removal of phenolsulphonphthalein has been investigated by comparing it to D_2O, whose exchange is known to be unaffected in hydrocephalus,[1,2] and by relating it to ventricular size. A comparison of phenolsulphonphthalein and D_2O exchange was made in 11 patients, 4 with communicating hydrocephalus and 7 with noncommunicating hydrocephalus. The time for half of the

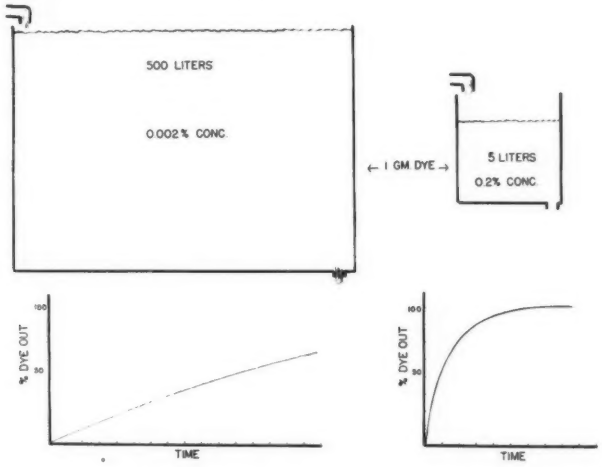

FIG. 2. This chart shows the effect of putting the same amount of dye into unequal volumes. The concentrations are different and so with equal flow the percentage removal of dye from the larger volume is much slower than from the smaller volume in which the concentration of dye is higher.

phenolsulphonphthalein or D_2O to exchange (half time) was used for comparison. The ratio of these two half times ($PSP^{T1/2}/D_2O^{T1/2}$) should be the same in both normal patients and hydrocephalic patients unless the rate of absorption of phenolsulphonphthalein is altered by the hydrocephalic state. If the rate of absorption of phenolsulphonphthalein has been decreased, its half time will be lengthened and the value of the ratio $PSP^{T1/2}/D_2O^{T1/2}$ will be increased. This, however, was not found to be the case. In patients with normal cerebrospinal fluid systems this ratio was found to be 31. The mean of this ratio in the 5 patients with communicating hydrocephalus was 24, and the mean in the patients with noncommunicating hydrocephalus was 30.

This is extremely good agreement for tests of this kind and it suggests very strongly that in hydrocephalus the absolute rate of absorption of phenolsulphonphthalein is not altered but it is only the dilution of the dye and the change in the surface/volume ratio of the ventricles that make this exchange apparently decrease. These half times are given in Table 1.

If the rate of absorption of phenolsulphonphthalein is dependent upon factors such as dilution and the surface of the ventricular wall, there should be a relationship between the absorption rate of the dye and the surface/volume ratio of the ventricle. It can be shown that the ventricular diameter can be used as a first approximation to this ratio* and that it should have linear relationship to the half time of phenolsulphonphthalein, the slope of this line being a measure of the absorption rate of phenolsulphonphthalein.* The ventricular diameter was measured from air studies of 100 patients; 50 had noncommunicating hydrocephalus and 50 had communicating hydrocephalus. There is indeed a linear relationship between the ventricular diameter and the half time of phenolsulphonphthalein excretion, as predicted. The regression lines for these two groups, calculated separately by the method of least squares, have the same slope, which indicates not only that there is a relationship between ventricular size and excretion of phenolsulphonphthalein, but also that it is the same for both communicating and

* The volume of a mass bounded by curved surfaces is a cubic function of the semi axes while the surface area bounding such a mass is a square. Thus the surface/volume ratio can be approximated r^2/r^3 or $1/r$. In the case of the lateral ventricles there are two so that the transverse diameter of one ventricle can be used to approximate the sum of the two radii.

PSP disappears from the ventricle as an exponential curve.

I $Csf = Co^{-xt}$ t = time after injection of the *PSP* into the ventricle.
Co = the maximum concentration of *PSP* in the *CSF* when $t = 0$.
Csf = the concentration of *PSP* in the *CSF* at any time "t".
x = a complex constant of exchange which includes the surface/volume ratio and the absolute exchange coefficient. For purposes of this paper it can be written S/VK.

Rearranging I and inserting $S/V\,K$ for x:

II $\ln Co/Csf = S/V\,Kt$
When $t = T1/2$ (the *PSP* half time in this case) $\ln Co/Csf = \ln 2$ by definition.
Placing this II and rearranging

III $\ln 2/S/V = KT1/2$
Substituting $1/d$ for S/V where d is the transverse diameter of the ventricle.

IV $d \ln 2 = KT1/2$
$\ln 2$ and K are constants, therefore; a plot of d vs $T1/2$ should be linear with slope $K/\ln 2$.

TABLE 1

Comparison of D_2O exchange and disappearance of phenolsulphonphthalein from ventricular cerebrospinal fluid in hydrocephalic infants

Cases	T1/2 PSP	T1/2 D_2O	Ratio PSP/D_2O
Expected normal			
Mean	5.2 hrs.	10 mins.	31
Communicating hydrocephalus			
D.P.	24 hrs.	95 mins.	15
D.W.	37 hrs.	78 mins.	28
S.K.	24 hrs.	80 mins.	28
D.W.	37 hrs.	81 mins.	24
Mean			24
Noncommunicating hydrocephalus			
A.L.	44 hrs.	75 mins.	35
E.P.	45 hrs.	178 mins.	15
K.C.	102 hrs.	155 mins.	39
C.C.	32 hrs.	80 mins.	24
M.T.	47 hrs.	147 mins.	19
S.W.	156 hrs.	173 mins.	60
M.T.	47 hrs.	147 mins.	19
Mean			30

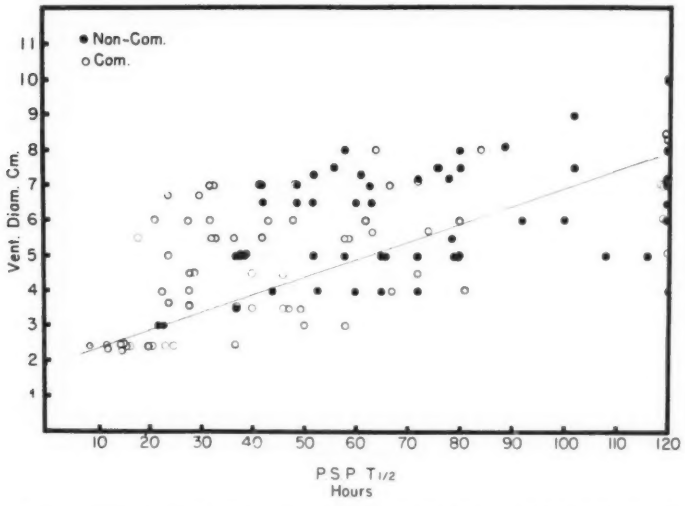

FIG. 3. A scattergram of ventricular diameter versus phenolsulphonphthalein half time. Open circles = communicating hydrocephalus. Solid circles = noncommunicating hydrocephalus. The slope of the regression line, which is a measure of phenolsulphonphthalein exchange, is the same for each group, calculated separately by the method of least squares.

noncommunicating hydrocephalus. There is considerable scatter of points, but that is not surprising when one considers that these data were taken from routine clinical procedures, in which the error of timing and collecting the specimens is always large. From the statistical point of view this great scatter of data makes the identical relationship found by calculation in these two large groups even more impressive, and the probability of it being chance is insignificant. The data for both groups, patients with communicating hydrocephalus and those with noncommunicating hydrocephalus, have been combined in Fig. 3.

Dandy and Blackfan[5] also measured the excretion of phenolsulphonphthalein when the dye was injected into the lumbar region. They found a decreased rate of excretion in communicating hydrocephalus, which they interpreted to mean that the pathways over the hemispheres were blocked. However, this decreased rate of urinary excretion was only the result of dilution of the dye from mixing with the large volume of ventricular fluid (which they showed occurred). Proof of this is furnished by their own animal experiments in which they ligated the spinal sac in the upper thoracic region, injected the phenolsulphonphthalein into the lumbar region and found normal rates of urinary excretion.

CONCLUSIONS

It seems from this analysis of these tests that absorption of phenolsulphonphthalein does not truly measure the state of the absorption mechanism of the cerebrospinal fluid, but measures only the increased volume of cerebrospinal fluid in a very complicated way. It has no real significance in assessing the clinical state of a hydrocephalic patient or in deciding what measures should be carried out. On the other hand, it is a very useful tool in testing for communication between the cerebral ventricles and the lumbar subarachnoid space, particularly when combined with adequate air studies.

SUMMARY

1. An evaluation of the use of phenolsulphonphthalein in the clinical investigation of hydrocephalic children has been carried out in 301 patients: 130 patients had communicating hydrocephalus and 137 patients had noncommunicating hydrocephalus.

2. Adequate communication probably exists between the cerebral ventricles and the lumbar subarachnoid space if dye appears in the lumbar cerebrospinal fluid within 10 minutes after being put into the ventricle when the test is carried out as described. Times of appearance longer than this are suggestive of an obstructive lesion, and if the dye does not appear within 18 minutes the patient probably has inadequate cerebrospinal pathways between the cerebral ventricles and the spinal subarachnoid space.

3. Adequate ventricular air studies should also be done, as some obstructive cystic lesions of the posterior fossa allow the dye to pass, but not a sufficient volume of fluid.

4. Any discrepancies between air studies and dye tests should indicate further study to confirm the diagnosis.

5. The absorption of phenolsulphonphthalein is not absolutely altered in hydrocephalus, but rather, its slow urinary excretion in hydrocephalus is the result of dilution in the enlarged ventricles; apparently this makes the absorption slow.

6. No clinical significance should be placed on slow absorption of phenolsulphonphthalein from the ventricles of a patient with hydrocephalus.

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SELECTIVE PARTIAL NEURECTOMY IN HEMIFACIAL SPASM AND THE ELECTROPHYSIOLOGIC SELECTION OF PATIENTS*

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WE ARE concerned here with the existence of hemifacial spasm as an apparent neurological or psychoneurological disturbance characterized by paroxysmal twitching of all, or part, of the musculature of the face. A review of the extensive literature on this subject has been presented by the authors[18] in another publication, and reveals a wide divergence of opinion regarding the etiology, classification, and treatment of the facial dyskinesias. Considering these discrepancies it seems strange that clinicians have not made more effective use of the aid proffered by electrophysiologic technics.

This discussion deals briefly with the assistance gained by the use of such technics and their practical applications.

Utilizing combined clinical and electrophysiologic observation, the hemifacial spasm occurring as an *isolated* neurological or psychoneurological entity can be classified as follows: (a) Spasm caused by cortical irritation. (b) Spasm resulting from involvement of the facial nucleus itself. (c) Spasm representing a psychogenic conversion reaction (habit spasm).

Hemifacial spasms occurring as an accessory symptom in a variety of neurological disorders (purulent meningitis, trigeminal neuralgia and tetanus,[3] encephalitis, [2,13] aneurysms of the basilar artery,[4] cerebellopontine tumors,[6] and following irritation of mes- and diencephalic areas in animals[12]) are not a topic of this discussion.

Hemifacial spasms caused by cortical irritation are somewhat uncommon since additional neurologic signs and symptoms resulting from involvement of adjacent cortical areas are usually present. In these cases actual disturbance of the cerebral neuronal rhythmicity is noted in electroencephalographic studies.

The cases of prime interest to the neurological surgeon, however, are those in which the spasms are caused by *involvement of the facial nucleus* itself, which Wartenberg[17] and others[8] believe to be the site of lesion in this condition. Wartenberg classifies hemifacial spasm caused by nuclear firing as (1) cryptogenic facial spasm resulting from infectious or degenerative changes, or from an inferior *Anlage;* (2) *réaction à distance* in lesions of the peripheral branches of the facial nerve; and (3) postparalytic facial spasm following

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Bell's palsy. It is *this* type of hemifacial spasm, i.e., caused by nuclear firing, which can, and necessarily must, be differentiated from the psychogenic variety of habit spasm if neurosurgical measures are to be considered.

Unlike Thiébaut *et al*[16] we have been unable to differentiate these various subtypes of firing electrophysiologically. It seems that there may be a common electrophysiological basis for the hemifacial spasm caused by nuclear firing. While these three subclassifications show no definite electroencephalographic disturbance of cerebral rhythmicity, all show an electromyographic

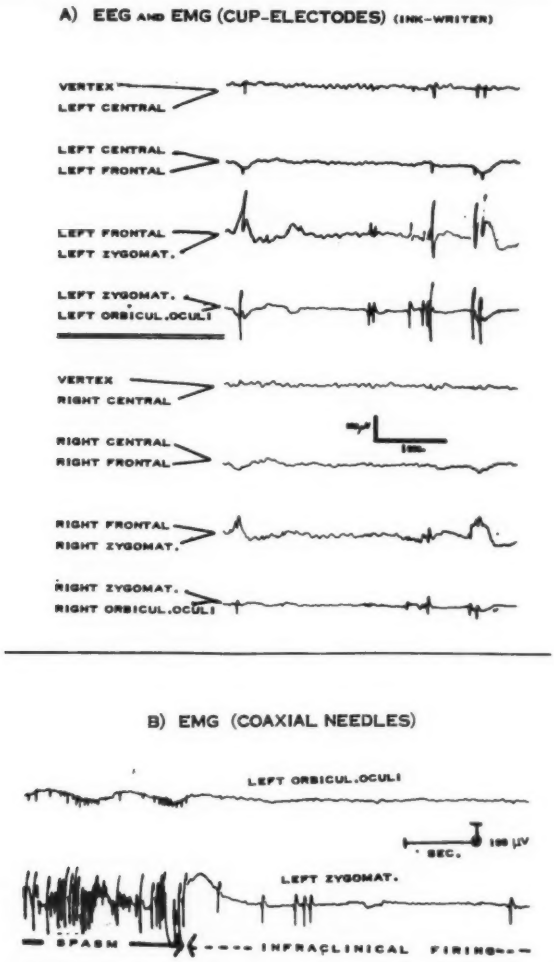

FIG. 1. Hemifacial spasm caused by "nuclear firing." Maximal mono- and biphasic spike bursts over left zygomatic muscle (in spite of clinical accentuation of spasms over left orbicularis oculi muscle). (A) Utilizing cup electrodes. (B) Utilizing coaxial needles (see text).

pattern of characteristic type. It was interesting to note that mono- and biphasic spike bursts firing in unison over the facial muscles showed maximal gain (200 μV) over the zygomatic muscle, while the visually apparent maximum twitching occurred in the left orbicularis oculi muscle. Occasionally single spike forms were noted firing in repetitive fashion. A polyphasic spike form was noted when recordings were made with coaxial needles placed in the zygomatic muscle. The characteristic electromyogram as seen in nuclear firing is presented in Fig. 1, demonstrating the typical fasciculation potentials recorded by a sensitive ink-writer apparatus. For adequate recording of the very much faster fibrillations which were observed by us and others,[7] particularly in the post-paralytic type, a single or preferably double-beam cathode-ray oscilloscope will have to be used.

This type of "nuclear" hemifacial spasm must be strictly differentiated from the *psychogenic* "habit" spasm which occurred much more frequently in our series of patients and which is characterized electrophysiologically by typical "contraction-bursts," lasting from 300–500 msec. (Fig. 2), and showing 5–7.5 rapid muscle spindles/100 msec.

Having used an electrophysiological approach first to determine if the hemifacial spasm is caused by (a) cortical irritation, (b) involvement of the nucleus itself, or is (c) actually a psychogenic conversion reaction (habit spasm), the problem of adequate therapy comes to mind.

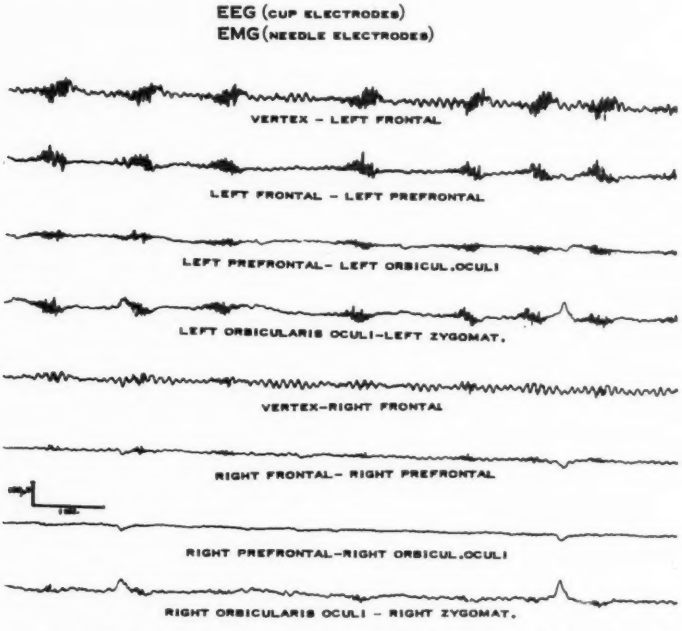

FIG. 2. Psychogenic "habit" spasm. Rapid contraction bursts over all left facial electrode derivations, each burst showing 5–7.5 muscle spindles/100 msec. (see text).

It is important, of course, that conversion reaction or spasm from cortical irritation be eliminated from any surgical consideration involving the 7th nerve. The obvious attack must be made upon this structure when the spasm is caused by nuclear involvement. Without destruction of all or part of the facial nerve, no relief of the hemifacial spasm is obtainable. Wartenberg[17] stated that only when the facial nerve is eliminated is the patient free of spasm. Numerous attempts have been made to relieve the patient, attempts ranging from freezing, Novocain, and/or alcohol injection, neurolysis, decompression and neurolysis, to stretching of the facial nerve.[19] Section with immediate resuture of the nerve was also recommended by Coleman,[5] as was changing the innervation of the involved muscles and anastomosing the facial nerve with another motor cranial nerve. The nerve-anastomoses reported included a facial-accessory anastomosis by Kennedy[15] and a facial-hypoglossal anastomosis by Gibson.[10] Simultaneous section of the cervical sympathetic chain to produce a Horner's syndrome with droop of the upper lid and some recession of the eye, with a narrowing of the palpebral fissure, has also been recommended.[1,14] However, in the presence of a bilateral hemifacial spasm the use of some of the other motor-cranial nerves would interfere seriously with eating and drinking.

German[9] proposed the isolation of several primary branches which, upon stimulation, caused contraction of those muscles particularly involved in the facial spasm. He recommended a small nerve flap composed of three-quarters or more of each of the involved branches, the free proximal segment to be held loosely in a reverse direction by a lightly tied suture. In 1946 Greenwood[11] proposed (in addition to the alcohol block of the nerve) an operative technic which sections the zygomatic branch completely as well as the lowest (cervical) branch, after repeated testing of the entire nerve by electrostimulation. When the facial nerve was separated into only two divisions, a partial division was done. By first dividing the nerve longitudinally with the point of a scalpel, an anterior section followed, much in the fashion recommended by German,[9] though the suture was felt to be unnecessary.

The procedure presented here differs from the others only in that it is a more selective partial neurectomy of the facial nerve. The principal fibers sectioned are those that are efferent to the predominantly affected muscles. Selection of these muscles is, of course, done by the previously mentioned electrophysiological technic, since the site of greatest clinical disturbance does not always correspond with the site of maximal electromyographic disturbance. Having determined which muscle group plays the greatest part in the production of the clinical picture, it is important that the greatest number of fibers to this particular zone be carefully divided, while others to less involved zones are more appropriately spared.

TECHNIC OF OPERATION

Under Pentothal anesthesia, with an intratracheal tube in place, the patient is prepared and draped for a preauricular operation. The drapes are so arranged that they may be elevated in order to observe the patient while stimulation studies are

being conducted. A curvilinear incision is made bordering on and paralleling the tragus of the ear, swinging below it and down the neck for a short distance (Fig. 3). The skin flap is then reflected forward, exposing the parotid gland. With stimulation studies the approximate location of the nerve within the gland is made and the gland is incised, usually near its anterior edge. The section is carefully carried down to the trunk of the facial nerve which is then exposed in its major divisions. Care must be taken to avoid damage to the parotid duct. When the facial nerve and its primary divisions have been isolated, attention is then directed to those branches supplying the area most severely involved.

Before any procedure is carried out on these branches, however, the entire facial nerve is stimulated, using a current of 2 to 3 volts of 60-cycle alternating current, usually of a single stimulus type. In keeping with Greenwood's[11] statement, the key to successful operation is the ability to locate the nerve and its branches, using electrical currents, and to gage the amount of weakness produced in the various muscle groups so that a noticeable paralysis is not produced. It is important that the orbicularis oculi be left with enough strength to close the eye and still eliminate most, if not all, of the twitching.

Having determined the amount of current necessary to stimulate the entire face on the affected side, the surgical attack on the individual divisions is made. Using the stimulating electrode to locate the branch leading to that muscle most seriously incriminated by the electromyogram, the individual nerve fibers are then teased free of the epineurium, using the dissecting needles (Fig. 4). After one such fiber is isolated over the needle point, the stimulator is applied. If the end-response to this stimulation is properly located with reference to the electromyogram, the fiber is then divided. The entire branch is then picked up and stimulated. Another isolated fiber is picked up, stimulated and divided. This procedure is continued until enough fibers have been sectioned in a specific nerve division so that only a limited response

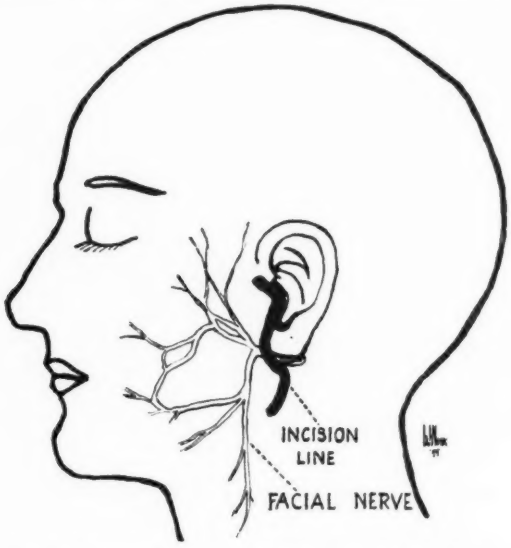

FIG. 3. Placement of incision for selective partial neurectomy.

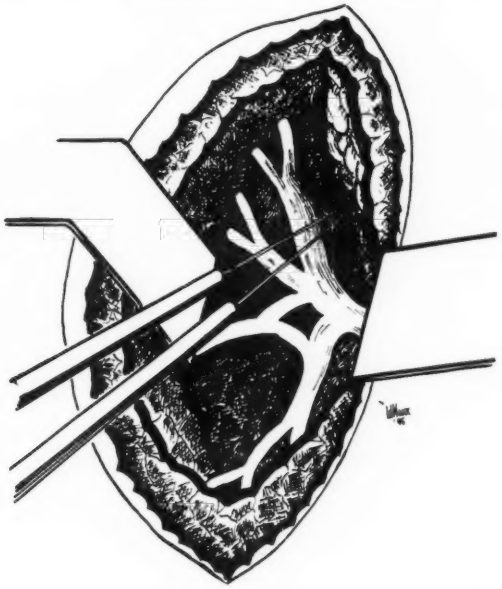

FIG. 4. Isolation of facial nerve fibers over dissecting needles (prior to stimulation and section).

in the innervated musculature results. Care should be taken to stimulate the entire nerve on completing this differential section, to determine the over-all response. Using the same current that was used to stimulate the nerve at the beginning of the operation, a considerably weaker response should be elicited. This procedure is followed down through the various divisions, different numbers of fibers being sectioned in each, consistent with the electromyographic response determined previously. Upon completion of this partial neurectomy in the involved divisions, the total nerve is again stimulated to note the over-all effect. Usually no further section is necessary at this point. The parotid gland is then resutured and the incision is closed in the usual manner, a pressure dressing being applied.

Upon awakening, the patient usually shows some weakness when smiling, though the resting face appears normal. While blinking of the eye is possible, it is somewhat sluggish. It is notable that after the 3rd or 4th day, postoperatively, an additional degree of paresis of the face occurs frequently, then subsequently begins to clear. When healing is completed, in the final evaluation of the procedure there is no evidence of paresis of the resting face, though upon smiling broadly slight weakness is apparent. Blinking motions of the eye are very nearly normal. It must be stated that occasional flicker-like spasms are still detectable, especially in oblique light, which are not at all disturbing to the patient.

Of particular interest here is the fact that most of the fibers to the more seriously affected part are divided, wherein this particular form of partial facial neurectomy differs from those previously employed. It is believed that

this more selective type of partial neurectomy is more satisfactory, not only from the functional, but from the aesthetic standpoint as well.

CONCLUSIONS

(1) Using electrophysiologic technics it is possible to isolate those cases of hemifacial spasm caused by involvement of the facial nucleus, or nerve radix, from those resulting from cortical irritation, or representing a psychogenic conversion reaction.

(2) At present the only successful approach for relieving the patient with hemifacial spasm caused by "nuclear and/or radicular firing" is a direct attack of varying degree upon the facial nerve itself, i.e., the executor of the spasm.

(3) The selective partial neurectomy wherein the fibers efferent to the site of maximum electromyographic disturbance are sectioned appears to be a more satisfactory approach to this particular surgical problem from both a functional and an aesthetic viewpoint.

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BENIGN TUMORS AT THE FORAMEN MAGNUM:

SURGICAL CONSIDERATIONS*

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AT TIMES, the symptoms of tumors affecting the spinal cord may resemble those of degenerative diseases of the central nervous system. This is ascribable especially, it seems to us, to the capricious nature of the unfolding symptoms of benign tumors in the vicinity of the foramen magnum. Unless the physician obtains an accurate history and attributes sufficient significance to the patient's often bizarre and at times seemingly functional subjective complaints, he is likely to deny the patient his ultimate opportunity for cure.

In a previous communication,[4] two of us (J.G.L. and H.W.D.) with Thelen pointed out that approximately 30 per cent of the neoplasms at the spinocranial junction are benign, extramedullary, and theoretically completely removable. In spite of the benignancy of tumors in this location the prognosis is influenced considerably by the size of the neoplasm and consequently by the duration of the symptoms caused by it. It seemed timely, therefore, to analyze the symptoms and signs of the tumor, and the clinical course in the 30 cases of benign tumors at the foramen magnum encountered at the Mayo Clinic from 1924 to 1956.

Twenty-six of these tumors were meningiomas and 4 were neurofibromas. Nineteen patients were females and 11 were males. The youngest patient was 19 years old, and the oldest 69 years. The age group from 40 to 50 years was the most common period for the occurrence of a benign tumor at the foramen magnum, 14 tumors being present in patients of this age (Fig. 1).

Twenty-seven of the 30 patients harboring benign neoplasms in this location underwent surgery. Of the 3 patients not operated on, 1, a woman in the last trimester of pregnancy, was moribund on admission and died shortly after admission; another elected to defer operation and died a few days later in respiratory failure, and the last patient was considered to have an inoperable lesion after pneumoventriculography. In each of these 3 cases, postmortem examination was performed, and the diagnosis was verified grossly and by microscopic section.

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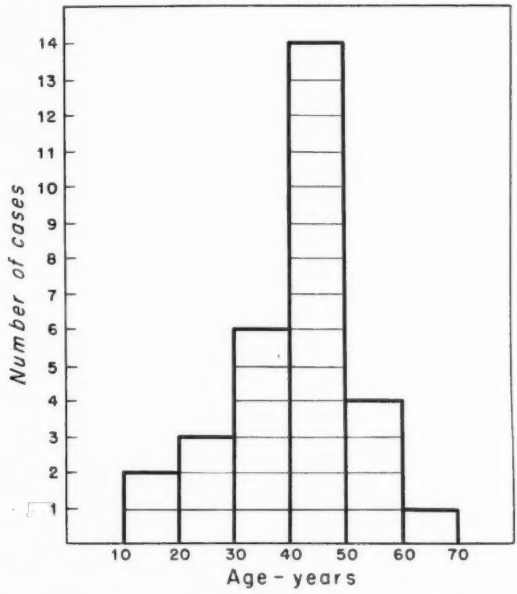

FIG. 1. Distribution of cases according to age of the 30 patients with benign extramedullary tumors affecting the neuraxis at the level of the foramen magnum.

SYMPTOMATOLOGY

The outstanding initial symptom in our series of cases was posterior cervical pain which frequently extended into the occiput. Fourteen patients gave cervical pain as the initial complaint and 6 gave suboccipital headache. Five additional patients said that one or the other of these symptoms had been present at some time. In a few instances cervical pain had preceded all other symptoms by several years, and in 1 instance, remarkably enough, by 12 years. The cervical pain was described as extending into the occiput and sometimes into the frontotemporal regions. The cervico-occipital pain was often influenced by coughing, straining, sneezing and the position of the patient's neck. Increasing discomfort was often initiated by movement of the neck, forcing the patient to keep his neck in a protective tilt.

In the course of the disease, paresthesia and definite numbness were frequently present in the cervico-occipital area, and hyperesthesia was not infrequent. In 2 instances the patient told of marked tenderness in the neck and occiput which rendered lying on his back almost impossible.

A feeling of weakness of an extremity, usually the upper extremity, was the next most common initial complaint; 6 patients mentioned this, while another 3 patients had paresthetic sensations in the hand or foot as the earliest symptom. In another patient, the initial symptom was low-back and sciatic pain; this was present for several years and was followed subsequently

by pain and numbness in the other lower extremity. This reminds one of the not infrequent occurrence of sciatic type of pain associated with tumors of the cervical portion of the spinal cord.

Paresthesia, described as tingling, prickling, burning and coldness, was present at some time in 26 patients and was seen in order of frequency in fingers, hands, arms, neck, scalp and legs. An interesting early complaint in 2 cases was tingling paresthesia starting in the fifth finger of one hand and gradually involving the fourth, third and second fingers, whereas the thumb was involved last. Subsequently, the other hand was involved in similar fashion.

A most intriguing finding among the sensory complaints was the influence of the patient's position in their production. In a typical case, the patient complained that his fingers became numb, and he experienced cramps in his arms only when he lay down. Another patient gave a similar history caused only by lying on his left side. Other symptoms were a "tight band" around the abdomen, neck, chest, or upper extremity or in all, electric shocks in the extremities on bending the neck, and tingling or numbness in the tongue. Because of girdle pain, abdominal discomfort, nausea and vomiting, one of our patients had cholecystectomy performed elsewhere.

Muscle weakness was present, at some time, in the history of 26 patients, although the neurologic examination did not always confirm this objectively. The weakness was commonly described by the patient as starting in one extremity, especially one arm, and then typically would progress to the lower extremity on the same side. Difficulty in swallowing was experienced by 9 patients, and respiratory difficulty or weakness of respiration was evidenced by 6 patients. A curious complaint of many patients was that the effort associated with ordinary conversation exhausted them. Because of this, on one or two occasions the diagnosis of myasthenia gravis was entertained early in the clinical evaluation.

One patient stated that the act of yawning was followed by a "drawing feeling" in his right extremities, followed by spontaneous purposeless movement of these extremities. Another patient complained that on sneezing or yawning, his extremities would become rigid and begin to shake.

Six patients were unable to stand at the time of admission, and 12 others gave histories of difficulty in walking. Several patients complained that they stumbled in the dark or had difficulty remaining on their feet on closing their eyes.

Although the severity of the symptoms described by the patients usually progressed steadily, on occasion, transient symptomatic improvement would follow contrast baths, vitamin therapy and the like. This often proved confusing in unraveling the true picture. Cervical pain, treated by manipulation, did respond favorably in a few instances. However, the immediate onset of transient quadriplegia in certain patients following such manipulation bespeaks the precarious existence on the precipitous edge of neurologic decompensation in which many of these patients exist.

NEUROLOGIC FINDINGS

Evidence of encroachment on the cranial nerves by these benign tumors was observed in 15 patients. In 9 patients the affection was bilateral, and in 6, unilateral.

The 11th cranial nerve was affected most commonly. This undoubtedly can be explained by its lateral location and extension of its roots of origin over three to four cervical segments. In 6 instances the disturbance was bilateral, and in 5 unilateral. Of the other cranial nerves, the 10th and the 12th were affected in 4 cases each, the 5th and 9th were affected in 3 cases each, and the 7th and 8th in 1 case each.

In 9 patients neurologic examination did not elicit any objective sensory changes. In another 11 patients disassociation occurred between the three sensory modalities. Frequently (7 cases) tactile sense was preserved, whereas the appreciation of pain and temperature was disturbed to varying degrees.

All modalities of sensation were disturbed on the entire body below the neck of 6 patients. In 1 patient (reported elsewhere[2]), the only remaining sensation was limited to the distribution on the face of the 5th cranial nerve, and even this was partially affected. In another 5 patients the sensory disturbance was entirely unilateral, as an arm and leg; in 4 hemianesthesia was found below the neck, and in another 4 anesthesia involved only one extremity.

Certain peculiar sensory patterns encountered in some of these patients were often the despair of the neurologist laboring to piece together the anamnesis and neurologic findings. One extremity might be spared a sensory deficit, or the sensory deficit might involve only the saddle area. Other deficits involved the homolateral hand and foot only, or both arms only. In 1 patient the sensory deficits were spotty, literally defying analysis. In 6 cases the most marked sensory disturbance was opposite to the side of the tumor. It should be mentioned, however, that the objective sensory changes at times were of such minor degree that they were difficult to elicit and record.

Loss of vibratory and joint sense did not always parallel the disturbance of other modalities of sensation, for sometimes these were preserved in the face of a disturbance of sensation of the entire body below the neck. An unusual finding was loss of vibratory and joint sense in the upper extremities and their preservation in the lower extremities.

Two patients showed no motor or sensory loss and 3 proved to have cranial nerve involvement only. In 7 no sensory deficit was elicitable, and only loss of motor power was found. This unusual state occurred in 3 patients who were suffering from severe loss of motor power. On the other hand, 1 patient had marked sensory loss without disturbance of motor function; 1 patient had all of the signs (motor and sensory loss, involvement of cranial nerve and Babinski response), but they were unilateral.

Of the entire 30 patients, only 3 had entirely normal muscle power. In general, if weakness was present, the upper extremities were weaker than the

lower extremities. The muscular weakness in the patients examined by us was always more pronounced on the side of the tumor. Four patients were quadriplegic and 5 others, quadriparetic. Seven patients had unilateral loss of motor function; 3 were hemiplegic, and 4, hemiparetic. Two patients had paraparesis of the upper extremities and 1, of the lower extremities. One patient had paresis of one arm, while only the small muscles of both hands were involved in 2 others. Involvement of the diaphragm occurred in 6 patients and proved to be a serious complication.

Reflex changes were present in all but 3 of the patients studied and the reflexes were, as a rule, hyperactive. The Babinski response was obtained in 25 patients, in 5 instances unilaterally.

A large majority (21) of the patients harboring benign tumors at the foramen magnum had evidence of a stiff neck or limited mobility of the neck, at times to a marked degree. This sign seems to us to be worthy of emphasis. Tenderness of the muscles of the neck on palpation and tenderness of the cervical vertebrae to percussion were also prominent signs in these patients.

Muscular co-ordination was recorded to be entirely normal in only 2 patients, and the Romberg sign was positive in 15 patients. Twenty-one of the 30 patients examined showed disturbances of gait frequently described as "spastic and ataxic."

OCULAR FINDINGS

About half of the patients afflicted with benign neoplasms at the level of the foramen magnum had horizontal nystagmus. Palsy of extra-ocular muscles occurred in 3 cases: it consisted of bilateral and unilateral 6th nerve palsy in 1 case each, and unilateral paralysis of the 3rd nerve in 1 case. In 1 other patient the corneal reflexes were decreased bilaterally.

One of the most interesting differences in this group of 30 benign tumors at the foramen magnum, as compared to our previous group of 74 tumors of all types, was the low incidence of choking of the optic disks associated with benign lesions (3, or 10 per cent of cases) and the high incidence of such choking (37 out of 74, or 50 per cent of cases) in the mixed tumor group. This suggests that the presence of papilledema in a patient with symptoms of a lesion at the level of the foramen magnum may be the clue for a guarded prognosis by the surgeon.

ROENTGENOLOGIC FINDINGS

In marked contrast to the mixed group of tumors in this area (previously reported), the benign tumors (neurofibromas and meningiomas) seen at the clinic showed surprisingly few roentgenologic changes. The outstanding roentgenographic change was enlargement of the intervertebral foramen between the 1st and 2nd cervical vertebrae (Fig. 2). At the clinic, routine roentgenographic examination of the skull includes postero-anterior, stereoscopic lateral, and Towne views. Anteroposterior, lateral and usually

FIG. 2. Marked enlargement of a cervical intervertebral foramen caused by benign extramedullary neurofibroma.

three-quarter views of the cervical portion of the spinal column also are obtained when a cervical lesion or a tumor at the foramen magnum is suspected.

DIFFERENTIAL DIAGNOSIS AND THE FINDINGS ON SURGICAL DIAGNOSTIC PROCEDURES

The presumptive diagnosis, which had been made on many of these patients before they were examined at the clinic, was multiple sclerosis, syringomyelia, amyotrophic lateral sclerosis, chronic encephalomyelitis or primary lateral sclerosis.

An additional group of diagnoses attached to patients afflicted with benign neoplasms of the foramen magnum, but less common, were: subluxation of the upper cervical vertebrae, brachial plexus neuritis, platybasia, protrusion of an upper cervical disk, cervical cord tumor and posterior fossa tumor.[1,5]

The pitfalls to early diagnosis are many, and at times it is difficult for the physician to readjust his frame of mind to suspect a slow-growing benign tumor in the vicinity of the foramen magnum once he has been lulled into

the positive security of the diagnosis of a degenerative disease of the nervous system beyond hope of recall.

After careful consideration of the laboratory and surgical tests employed as aids to the clinical neurologic diagnosis in these cases, certain recurring points of interest have come to attention.

In previous papers[3,4] the surgical diagnostic methods employed in visualization of the spinocranial junction, such as ventriculography, pneumoencephalography, and spinal myelography with radiopaque iodine, and the surgical philosophy behind the individual selection of these tests have been discussed and need not be mentioned further here (Figs. 3 and 4).

Of some importance, however, is the knowledge that in the presence of a benign tumor at the spinocranial junction, 5 of the 20 patients who underwent spinal puncture had complete blocking of the subarachnoid pathways of the spinal fluid and 6 others had partial blocking. The Queckenstedt maneuver was carried out in 19 cases. Papilledema was associated with only 3 of the 30 cases of benign tumors and when there is papilledema the Queckenstedt test should not be done.

FIG. 3. Complete spinal subarachnoid block to the passage of ethyl iodophenylundecylate (Pantopaque) by a benign tumor at the foramen magnum. Occasionally herniation of the cerebellar tonsils through the foramen magnum will cause a defect in the oil column which resembles the outline of a tumor.

The values for protein in the spinal fluid ranged from 55 mg. to 2,400 mg. per 100 cc. Oddly enough, the lowest value of protein in the spinal fluid and the highest obtained occurred in the presence of neurofibromas. The cellular content of the spinal fluid of 18 of the 20 patients undergoing spinal puncture was normal (Table 1).

Once again we believe it timely to recommend certain precautions when spinal puncture is undertaken in cases in which a lesion high in the cervical portion of the spinal canal is suspected. The surgeon should see that facilities are available not only for the maintenance of respiration if sudden respiratory failure should ensue, but also for immediate decompressive cervicooccipital laminectomy, if necessity dictates. Emergency ventricular tap and drainage might even be necessary to "uncork" the foramen magnum.

The electroencephalograph has proved of little value as a diagnostic aid, but more recently electromyographic studies have given much promise as a valuable laboratory method of more precise localization by dermatometric means.

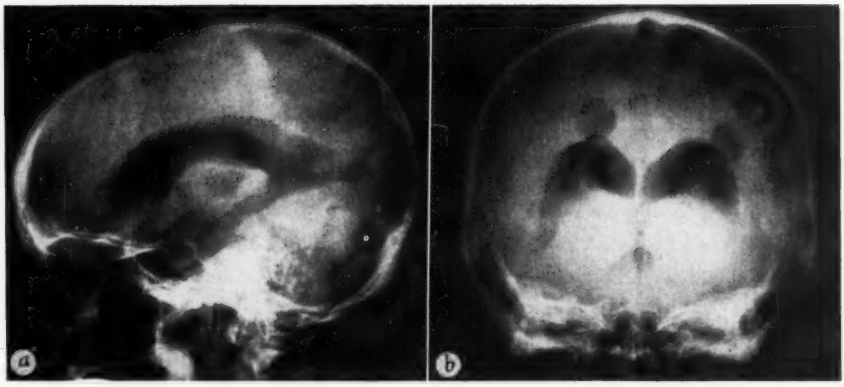

FIG. 4. Pneumoventriculogram to demonstrate distortion of lower end of aqueduct of Sylvius and the 4th ventricle by an anteriorly placed meningioma at the foramen magnum. (A) Lateral view. (B) Postero-anterior view.

On occasion the surgeon will be placed in the depressing situation of encountering a patient with the clinical picture of progressive "demyelinizing" disease of the upper cervical part of the cord on whom all special tests are of relatively little value, and who has a high normal protein content in the cerebrospinal fluid (40 mg. per 100 cc.). It is our belief that after frank and considerate explanation to the patient and his relatives of the many facets of the problem, a direct cervico-occipital exploration is preferable to a regimen of watchful waiting until neurologic deterioration precludes recovery should a remediable lesion be ultimately uncovered.

SURGICAL ASPECTS

The surgical attack on tumors situated at the foramen magnum has been previously considered in some detail but certain facts stand out con-

TABLE 1

Protein content of cerebrospinal fluid

Protein, mg. per 100 cc.		
Less than 100	100–200	More than 200
55*	110	350
70	120	400
80	120	950†
90	140*	2,400*†
	150†	
	150	
	150	
	160	

* A neurofibroma was associated.

† These figures denote a complete block to the subarachnoid pathways of spinal fluid. One additional patient had complete spinal block but protein values in the spinal fluid were not determined.

cerning the handling of benign extramedullary neoplasms in this region. Respiratory problems are frequent in these cases and also in cases in which swallowing is affected because of involvement of the 10th cranial nerve and adjacent nerves. Preoperative tracheotomy may be essential for the maintenance of an adequate airway and for better evacuation of secretions both during and after operation. During surgical intervention, the upright position is employed most commonly unless a definite contraindication exists. One of our patients ceased breathing every time he was placed erect, and another patient had to be operated on in a chest respirator. It should be emphasized that flexion of the neck may be hazardous in positioning patients with benign tumors at the foramen magnum for operation and may cause or add to existing respiratory embarrassment.

On occasion, artificial respiration had to be resorted to shortly after the beginning of the operation, and respiratory function was regained only when the dura mater over the foramen magnum was opened. On a few occasions, a respirator was kept ready postoperatively in order to assist the patient's breathing should it become necessary. That respiratory trouble can occur with a paucity of preoperative respiratory warning is well illustrated by the following case.

Case 1. A 42-year-old white housewife first registered at the clinic on Dec. 29, 1954, for a main complaint of pain in the left side of her neck extending up into her scalp over the back of her head. The patient stated that she had been in good health until approximately a year and 9 months before admission. At that time, April, 1953, pain began in the back of her neck on the left and extended to the back of her head on the same side. This pain became progressively worse. In the meantime, the patient had become pregnant.

In January, 1954, after delivery of her child, some remission of the pain took place for several months, but then it gradually worsened. Six months later she began to experience throbbing bandlike headache, which was more severe than the neck pain, which was still present. By September, 1954, weakness developed in the patient's left arm with loss of fine movements in the left hand, and she also noted a minimal tremor in this hand.

One month prior to admission at the clinic the patient noted that she was unable to judge the temperature of water with her right leg, and she also began to complain of numbness and "weakness" of the right leg. Weakness, however, was also noted in the left leg, even to a more severe degree. Continual stiffness of the neck bothered the patient considerably and, for the past several months, she had had difficulty in voiding.

Examination. General physical findings were unremarkable. Ophthalmologic examination revealed that the gross visual fields, extra-ocular movements and reactions of the pupils were normal. The optic disks were normal. The positive neurologic findings were Hoffmann's reflex on the left, and moderate weakness of the muscles of the left shoulder and upper extremity. The adductor, abductor, the hamstrings and the quadriceps group of the left lower extremity were weakened slightly. There was moderate decrease of touch, pain and temperature sense of the entire right side of the body below the chin; vibration and joint sense were normal. Co-ordination was impaired in the left extremities. The plantar response was flexor.

Results of routine laboratory studies of the blood and urine were within normal

limits, and roentgenograms of the skull, cervical vertebrae and chest showed normal conditions. A diagnosis was made of tumor high in the cervical portion of the spinal canal or possibly at the foramen magnum.

Examination of the spinal pathways after injection of ethyl iodophenylundecylate (Pantopaque) revealed a well-circumscribed filling defect in the oil column at the level of the 1st and 2nd cervical segments. Block of the spinal subarachnoid pathways to the Queckenstedt test was almost complete. During the lumbar puncture, clear colorless cerebrospinal fluid was obtained at an initial pressure of 6 cm. of water. The spinal fluid had a total protein content of 150 mg. per 100 cc. while 2 lymphocytes and 2 erythrocytes were present (gold 001110).

Operation. On Jan. 6, 1955, the patient was operated on in the upright position; thiopental (Pentothal) anesthesia was used. A laminectomy of the 1st and 2nd cervical vertebrae was performed and the rim of the foramen magnum was removed together with a small portion of occipital bone. Shortly after the beginning of the operative procedure, the patient's respirations had to be assisted. As soon as the rim of the foramen magnum was removed and the dura mater was opened, the patient's respirations improved markedly. A large meningioma was uncovered on the left side, anterior and lateral to the upper cervical portion of the spinal cord and to the medulla; it arose from the dura mater opposite the 1st cervical nerve root. It was necessary to sacrifice the 1st cervical nerve and a portion of the posterior root of the 2nd cervical nerve in order to remove the tumor from its lateral attachments. After removal of the tumor had been completed, the cord pulsated normally and it did not show any gross damage other than distortion caused by the tumor.

Postoperative course was uneventful. When last seen 10 months after operation, the patient appeared to be much improved. No weakness could be detected in the extremities. Deep tendon reflexes were moderately increased, but the plantar reflexes were bilaterally flexor. There were no sensory abnormalities except for the loss from section of nerve roots at operation and almost complete absence of joint sense in the left lower extremity, and mildly decreased joint sense in the left upper extremity.

Location of Tumors. Four tumors were found at the time of operation in a position lateral to the cervical part of the spinal cord and medulla, and 5 neoplasms were found readily in a posterolateral position. But more often, in 20 instances, the position of the benign tumor was anterior or anterolateral to the upper cervical part of the spinal cord and to the medulla.* On several occasions the cerebellar tonsils had herniated below the foramen magnum, sometimes to the level of the 2nd cervical vertebrae (Fig. 5).

With tumors arising in an anterior position (basilar groove), the upper cervical part of the cord and medulla were pushed posteriorly, rotated and stretched over the tumor, and often were thinned up to a third of the normal size (Fig. 6). On occasion, the tumor presented only with a small part projecting through the foramen magnum, making it possible to deliver the neoplasm without removal of the posterior rim of the foramen magnum. The 1st and 2nd cervical roots and the rootlets of the 11th nerve were often stretched over the tumor, when the latter was in an anterior position. Re-

* In 1 case in which the lesion was considered inoperable by us and the patient died elsewhere, the brain was returned for pathologic examination and study.

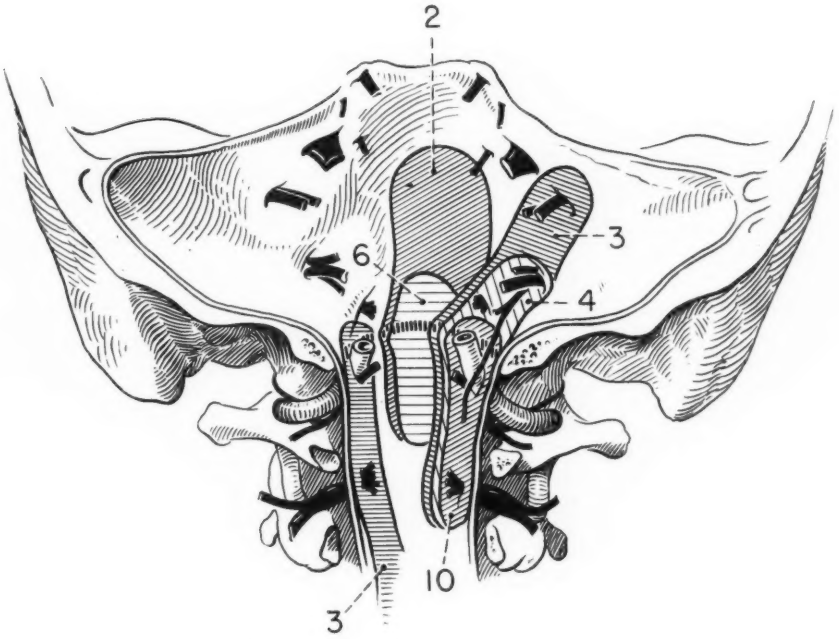

FIG. 5. Location of 28 of the 30 benign neoplasms occurring at the foramen magnum. One patient not included had multiple tumors, and another patient died elsewhere and the exact location at the foramen magnum could not be pathologically verified by us. The arabic figures indicate the number of times benign tumors were encountered in each location. The cranial and upper cervical nerves are shown in solid black.

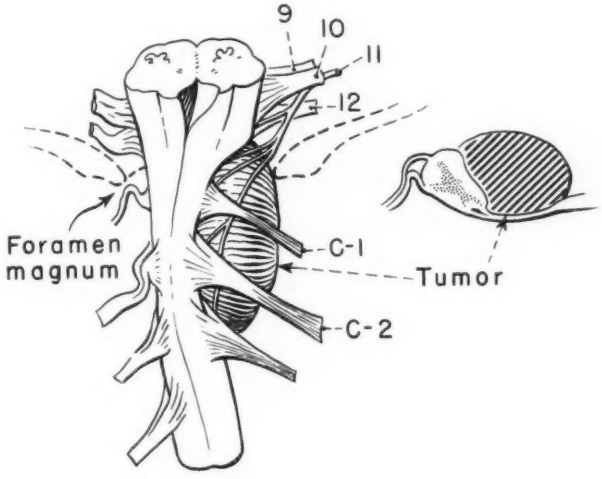

FIG. 6. Compression and rotation of the upper cervical spinal cord and medulla by a benign extramedullary tumor. Note the thinning of the cervical portion of the spinal cord and the stretching of the nerve roots.

peatedly, the 1st or 2nd cervical root or rootlets of the 11th nerve had to be divided to allow delivery of the tumor.

At times the vertebral artery was compressed by, or embedded in, tumor tissue or was stretched over the dome of a tumor. The basilar artery was occasionally involved in similar fashion. The 9th, 10th and 11th nerves and adjacent major blood vessels sometimes were embedded in tumor tissue, and on several occasions the neoplasm extended high into the cerebellopontine angle on the same side. Often small branches of the basilar or posterior inferior cerebellar artery contributed part of the blood supply of the neoplasm. The tumors originated from the 1st or 2nd cervical nerve roots, the upper cervical part of the dura mater, the rim of the foramen magnum, around the jugular foramen, and in the basilar groove anterior to the medulla. At other times, however, it was impossible to determine the exact origin of the neoplasm.

When the bulk of the neoplasm lies anterior to the cervical portion of the spinal cord or to the medulla, bilateral section of the dentate ligaments and anterior and posterior cervical nerve roots, if necessary, allows greater mobility. The tumor should frequently be approached and removed piece by piece with utmost care before removal of the capsule is attempted, and the capsule or the attachment of the growth to the dura mater may have to be left behind when it is located anterior to the cord or in the basilar groove, or encroaches on the basilar or vertebral artery. An extension of the tumor into the cerebellopontine angle may so encroach on the contiguous cranial nerves that complete removal is difficult or fraught with too great a hazard. It should be realized that complete removal of tumors in this anterior (basilar groove) or anterolateral position, despite their benignancy, may yield only a Pyrrhic victory gained at the cost of great damage to cord or medulla, or ultimate demise of the patient.

Sixteen tumors were classified as spinocranial and 14 as craniospinal. Of the 26 meningiomas in this series, 3 were classified pathologically as cellular

TABLE 2

Results of follow-up inquiries and re-examination of 30 patients

Results	Cases
No operation	3
Operative deaths	2
Lost to follow-up	4
Postoperative results	
Died in 3 or 4 months	2
Died 2, 5, 17 and 19 years after operation of other causes; neurologically well	4
Alive and well*	12
Alive but has neurologic deficit 2, 2 and 6 years after operation	3
Total	30

* Time since operation: 4 to 10 months in 4 cases; 2 years in 3 cases; 7 years in 2 cases; 3, 4 and 20 years in 1 case each.

active meningiomas. One patient died 10 hours after operation and another, 7 days after operation of medullary infarction. Four patients were lost to follow-up. Results in the remaining cases are given in Table 2.

A striking immediate result was the rapid and often unexpected neurologic improvement in many cases once the benign extramedullary compressive lesion had been removed.

COMMENT

As has been brought out by ourselves and others, the difficult discovery and the bizarre clinical patterns associated with benign extramedullary neoplasms in the vicinity of the foramen magnum, particularly those in an anterior or basilar groove location, may be ascribable to several factors.

In the first place, the cerebrospinal fluid cisterns of this region allow room for steady continued growth and displacement of adjacent neurologic structures to a sometimes marked degree before the limit of their tolerance is reached or before the continuing pressure of the growing tumor causes impingement of the adjacent neural tissue against some hard unyielding surface. In addition, a major portion of the blood supply to the upper cervical part of the cord and to the medulla, as the vessels lie anteriorly, may become progressively compromised and may give peculiar unrelated and distant bizarre signs, difficult to place in a diagnostic pattern. Moreover, obstruction to the cerebrospinal fluid pathways by the extramedullary masses may not give the helpful warning signs of headache, nausea, vomiting and papilledema until late in the course of the disease because of the room afforded by the subarachnoid cisterns in this area. The common occurrence of cervico-occipital pain, stiff neck and paralysis of the 11th cranial nerve are all readily explainable by the adjacent anatomy.

Are there, then, any signposts associated with these benign tumors that can put the physician on guard early? In the analysis of our 30 cases of benign tumors, it has occurred to us that despite the classic picture of a degenerative disease of the nervous system, the clues described in Table 3 should alert the physician to the possibility of tumor.

Too many patients in our series were given an initial diagnosis of degenerative disease of the central nervous system, especially in the early stages of the disease, when surgical attack could be most successful. Paresthesia and indefinite sensory disturbances are the pitfalls that contributed to the fact that multiple sclerosis was the most common misdiagnosis. This occurred often in our series. The resemblance to disseminated demyelinizing disease may be most striking: paresthesia, bizarre sensory disturbances, in-co-ordination, spastic ataxic gait, nystagmus, tremor and disturbance of sphincter function are common both to tumors at the foramen magnum and multiple sclerosis.

Unilaterality of sensory or motor disturbances, absence or unilaterality of involvement of cranial nerves, absence of pallor of the optic nerve heads or sheathing of the retinal blood vessels, and age of the patient may serve

TABLE 3

The most frequent symptoms and signs associated with 30 benign tumors of the foramen magnum

Symptoms
1. Posterior cervical or cervico-occipital pain.
2. Muscle weakness, in upper extremities initially, then progressing to lower extremities.
3. Unexplained paresthesia in upper extremities initially.
4. Subjective sensory disturbances, often bizarre in pattern.
Signs
1. Stiffness of the neck with aggravation of symptoms by certain positions of the neck.
2. Muscle weakness and signs referable to pyramidal tract.
3. Objective sensory loss, possibly without anatomic pattern.
4. In-co-ordination and disturbance of gait out of proportion to other neurologic disability.
5. Involvement of cranial nerves, especially the 11th.

at times to suggest the correct diagnosis. The most important single symptom of tumor of the foramen magnum is cervico-occipital pain. This pain is frequently caused by encroachment of the tumor on the 2nd cervical nerve root. Aggravation of this pain by motion of the neck and straining and a protective tilt of the head can often be observed. We feel justified in emphasizing that any patient who complains of cervical or cervico-occipital pain or stiffness, and has in addition signs and symptoms that may be considered typical of demyelinizing disease deserves careful re-evaluation and further study before quadriplegia and respiratory failure are allowed to develop.

Often despite much clinical zeal and scientific ardor, the correct answer, a benign anterior (basilar groove) tumor, can be obtained only if the diagnostic regimen is all-encompassing and includes giving the patient every diagnostic opportunity to reveal a tumor. Such a regimen may even have to include direct surgical exploration before the possibility of tumor is discarded and the diagnosis of demyelinizing disease is accepted. In dealing with the diagnostic dilemmas presented by benign extramedullary neoplasms affecting the upper cervical portion of the spinal cord and the medulla in the foramen magnum, clinical suspicion is hardly enough. It must be generously supplemented with a large dose of surgical diagnostic endeavor!

SUMMARY

Benign extramedullary tumors, arising at the level of the foramen magnum, have been encountered in 30 cases at the Mayo Clinic between the years 1924 and 1956. The initial diagnosis of these tumors was often "degenerative disease" of the nervous system. The significant symptoms, neurologic signs and pertinent, contributory laboratory studies are considered in detail. Posterior cervical pain, cervico-occipital headache and nuchal stiffness were present frequently and were of diagnostic importance. The information to be obtained from examination of the cerebrospinal fluid and

special surgical diagnostic tests is discussed. Roentgenologic changes in the skull and upper cervical vertebrae associated with these benign extramedullary neoplasms are few, but an important roentgenologic sign may be the enlargement of the foramen between the 1st and 2nd cervical vertebrae. Respiratory complications are frequent and a method of surgically treating patients harboring these tumors is discussed. Despite considerable diagnostic effort because of the often complex and bizarre symptoms and signs, the physician to establish the correct diagnosis must be possessed not only with a high index of tumor suspicion, but also fortified with considerable surgical diagnostic endeavor. The end results justify the unusual mental and physical effort required to handle these patients successfully and amply reward the patient and surgeon.

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THE RESULTS OF SURGICAL TREATMENT OF INTRACRANIAL ANEURYSMS AS DEMONSTRATED BY PROGRESS ARTERIOGRAPHY*

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IN ORDER to determine the results of surgical therapy for intracranial aneurysms, postoperative arteriography has been done as a routine procedure in patients having ligation of the carotid arteries in the neck and more recently in patients who have had a direct surgical attack on the aneurysm intracranially. The main purpose was to determine whether the artery was actually occluded and whether the aneurysmal sac was or was not visible. The means of establishment of collateral circulation is an interesting side-light which is being published in another paper.

The procedure followed in progress arteriography is to inject the artery proximal to the site of ligation whenever this is possible. This is not possible when the common carotid artery is ligated, as this artery is most often thrombosed proximal to the site of ligation and actually distal to the ligation as far superiorly as its bifurcation into the external and internal carotid arteries, and no pulsation of the common carotid artery is felt. If pulsation of the common carotid artery is palpable the artery is likely not completely ligated. In 2 cases of common carotid artery ligation the internal carotid artery was injected: in 1 case, by the percutaneous method and in 1, by the open method. When the internal carotid artery has been ligated or the intracranial approach has been used, the common carotid artery is injected, and the status of the aneurysm and of the clamp may be determined in some cases with a single injection. In the case of internal carotid ligation, the carotid syphon and the middle cerebral artery may be well shown by back-flow through the ophthalmic artery. If the artery at the site of the aneurysm is not shown, another arteriogram is done by injection of the opposite carotid. If still the site of the aneurysm is not shown, a vertebral arteriogram is done. In some cases it was not possible to demonstrate the site of the aneurysm.

Fifty-four progress arteriograms were performed in 51 cases at an average time of $10\frac{1}{4}$ months postoperatively, the shortest period being 10 days and the longest being 7 years and 10 months. Actually, a greater number of progress arteriograms were done, but only the latest postoperative angiograms were included in this study.

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TABLE 1

Case material and therapy

Location of Aneurysm	Therapy		
	Ligation Internal Carotid	Ligation Common Carotid	Direct Intracranial Approach
Internal carotid	22	7	6
Middle cerebral	6	1	
Anterior communicating		1	6
Anterior cerebral			1
Posterior cerebral			1

Of these 51 patients, 35 had aneurysms on the internal carotid artery; 6, on the middle cerebral, 1 of whom also had an internal carotid aneurysm on the same side; 7, on the anterior communicating; 1, on the anterior cerebral; and 1, on the posterior cerebral (Table 1). There were 4 patients who had multiple aneurysms: in 1 case both aneurysms were on one side of the brain, and in 3 cases they were on both sides. The 3 patients in whom the aneurysms were located bilaterally had no therapy directed to the aneurysms on the second side. Thirty-three patients had had subarachnoid hemorrhage. Ligation of the internal carotid artery in the neck was performed in 30 cases. Eleven patients had common carotid ligation, 3 of whom previously had had unsuccessful ligation of the internal carotid, using a tantalum clip. Thirteen had intracranial surgical therapy for the aneurysm. Some of the patients had more than one surgical procedure when the first operation was found to be ineffective, and progress arteriography was done after each procedure.

There were 43 progress arteriograms in which the artery was demonstrated at the site where the aneurysm arose. This, of course, was not possible in the 5 cases in which trapping was done by ligation of the internal carotid artery in the neck and intracranially, and in most of the cases in which ligation of the common carotid was performed. Of these 43 cases, the aneurysm was not seen on the angiogram in 24, although its site was well demonstrated; it was smaller in 9; unchanged in 8; and larger in 2. Further explanation, however, is necessary since included in the 43 cases are those in which the clamp or clip was found to be open and also those in which the clamp had to be released because of insufficient collateral circulation; in other words, those cases in which surgical therapy was incomplete.

The most significant cases are those in which progress arteriography showed both the site of the aneurysm and the clip or clamp to be closed (Table 2). There were 10 such patients who had an aneurysm of the internal carotid artery and ligation of the internal carotid artery. In 8 of these 10, the aneurysm was no longer visualized and in 2 it was the same size. In 4 such patients who had an aneurysm of the middle cerebral artery and who were shown to have complete ligation of the internal carotid artery, the aneurysm was not shown in 1 and was smaller in 3.

TABLE 2

Cases in which progress arteriogram showed site of aneurysm and showed that the clamp or clip on internal carotid artery remained closed

Location of Aneurysm	Aneurysm not Shown	Aneurysm Smaller	Aneurysm Same	Aneurysm Larger
Internal carotid	8		2	
Middle cerebral	1	3		

TABLE 3

Cases in which progress arteriogram showed site of aneurysm and showed that the clamp or clip on internal carotid artery was open

Location of Aneurysm	Aneurysm not Shown	Aneurysm Smaller	Aneurysm Same	Aneurysm Larger
Internal carotid	2	4	1	1
Middle cerebral			1	

The progress arteriogram showed the aneurysmal site and showed the clamp or clip to be open on the internal carotid artery in 9 cases (Table 3). In 8 of these cases the aneurysm was on the internal carotid artery. It was not seen in 2 cases, it was smaller in 4, larger in 1, and the same size in 1 case. There was 1 case in which the aneurysm was on the middle cerebral artery and in this case the aneurysm was the same size.

There were 5 cases in which the aneurysmal site was shown and the clamp or clip on the internal carotid artery had been released because of insufficient collateral circulation (Table 4). In 4 of these cases, the aneurysm was on the internal carotid artery; in 2 of these, the aneurysm was not visualized and in the other 2 it was the same size. In 1 case the aneurysm was on the middle cerebral artery and the size of the aneurysm was unchanged.

The aneurysmal site was shown in the progress arteriogram of 9 patients who had had common carotid artery ligation. In only 1 case could we demonstrate that the clip was closed. In this patient, the aneurysm on the internal carotid artery was not seen. In 2 cases the clamp had to be released;

TABLE 4

Cases in which progress arteriogram showed site of aneurysm in patients whose clamp or clip on internal carotid had to be released because of insufficient collateral circulation

Location of Aneurysm	Aneurysm not Shown	Aneurysm Smaller	Aneurysm Same	Aneurysm Larger
Internal carotid	2		2	
Middle cerebral			1	

in 1, the aneurysm, on the internal carotid artery, was found to be smaller, and in the other, the aneurysm, on the anterior communicating artery, was found to be the same. In 6 cases the status of the clamp could not be determined; in 2 of these the aneurysm on the internal carotid artery was not seen. in 1 case the aneurysm on the internal carotid artery was smaller, in 2 others, the aneurysm on the internal carotid artery was the same. In 1 case of middle cerebral aneurysm the sac was the same size in the progress arteriogram.

There were 13 arteriograms performed on patients who had had intracranial therapy for their aneurysms. Five of these had trapping by placing a clamp or clip on the internal carotid artery in the neck and intracranially, and the intracranial clip prevented the aneurysmal site from showing. Four had trapping intracranially by clips on the artery on either side of the site of origin of the aneurysm. In 3 of these, the aneurysm was not shown although the artery filled well up to the clip. In 1, the clips had become loos-

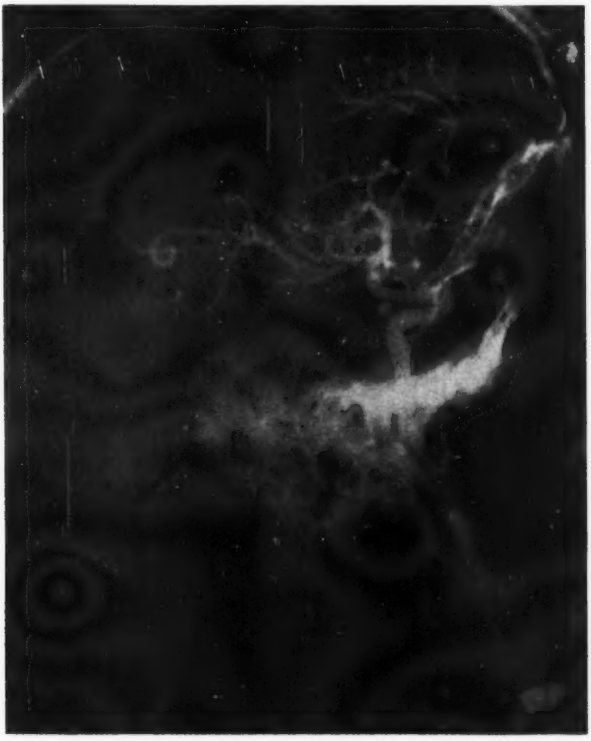

FIG. 1. Arteriogram showing Diodrast passing through internal carotid artery at site of the tantalum clip on the artery, and revealing that the aneurysm is present on the internal carotid artery, although it is smaller than it was before ligation.

ened and dislodged, and the aneurysm was the same size it had been before therapy. Two patients had the lumen of the aneurysm packed with muscle and in neither was the aneurysm shown. In the 1 patient who had muscle packed around an anterior communicating aneurysm, the progress arteriogram $3\frac{1}{2}$ months later showed the aneurysm to be the same size but different in shape.

We have a control group of 16 patients who had intracranial aneurysms demonstrated arteriographically and who subsequently, without any surgical therapy, had a second arteriogram. The time between the first and the second arteriogram ranged between 2 weeks and 15 months. The average time was 3 months. In 13, the aneurysm was the same size and in 3, it was larger.

Some of the interesting facts are the following: there were 8 patients who had a tantalum clip used for the ligation. In 6 of these, the clip was open (Fig. 1), in 1 the clip was closed on the common carotid artery, and in the other case, the internal carotid artery was clipped both in the neck and intra-

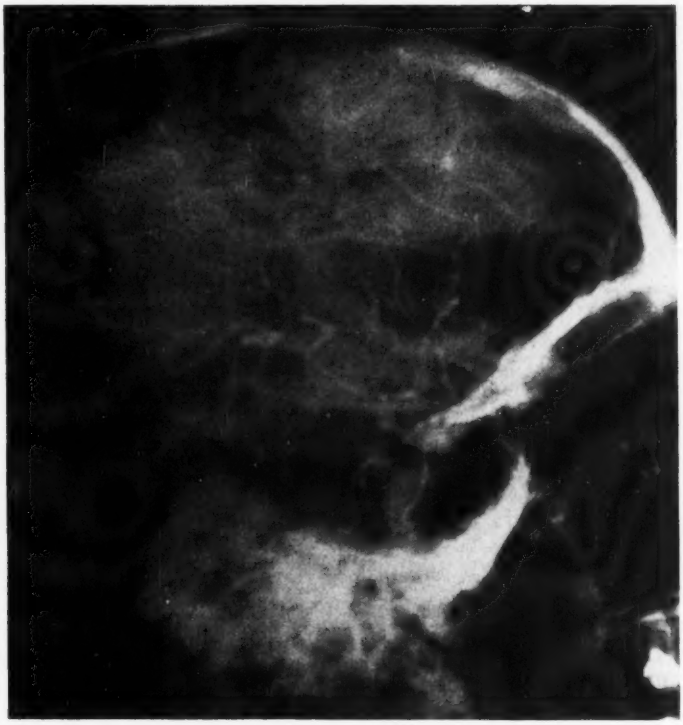

FIG. 2. Pre-operative arteriogram showing aneurysm of internal carotid artery in a patient who later had the internal carotid artery ligated. Hemiplegia developed, from which the patient did not recover completely when the clamp was opened.

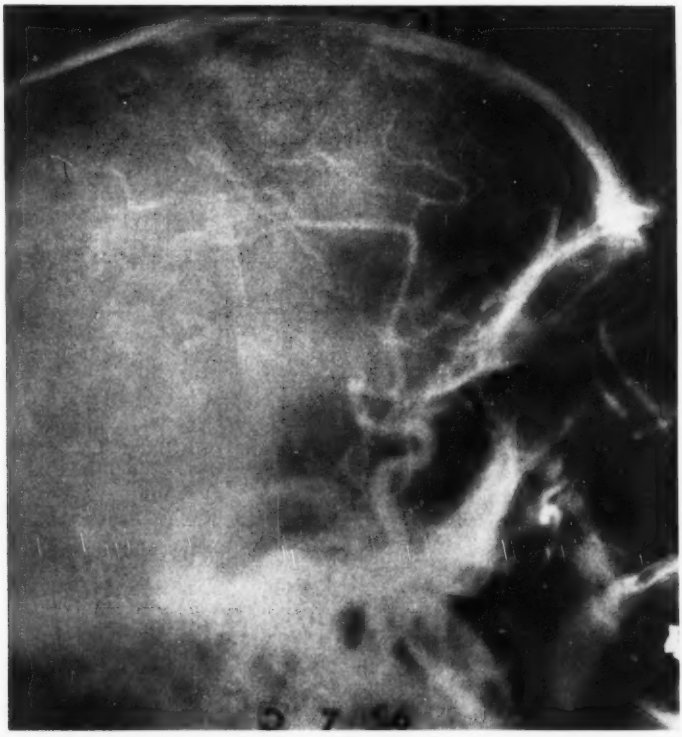

FIG. 3. Progress arteriogram (same case as in Fig. 2) shows incomplete thrombosis of middle cerebral artery; only two small thread-like arteries remain. There is a small defect in the internal carotid artery which represents the residual neck of the aneurysm.

cranially. In 1 case the artery had recanalized around one side of the clip. In using the Selverstone clamp it is necessary to do a final firm tightening of the clamp before removal of the tools, otherwise the artery will not be occluded. This was found to be true in 5 of our cases. In 1 such patient the progress arteriogram showed the clamp on the internal carotid artery to be open and the aneurysm was smaller. This patient had no complications when the clamp was thought to be closed, but the second time the clamp was closed evidence of insufficient collateral circulation developed 8 hours later. This suggests that the clamp was not closed completely on the first attempt. There were 2 patients who had a hemiplegia after arteriography, from which they did not completely recover upon prompt opening of the clamp. In both of these patients, thrombosis of the middle cerebral artery was found when the progress arteriograms were done (Figs. 2 and 3). In 1 patient who had an aneurysm of the middle cerebral artery and was treated by ligation of the internal carotid artery, the progress arteriogram showed

that the Selverstone clamp was not completely occluding the artery and the aneurysm was the same size. The clamp was closed completely and another arteriogram 2 months later showed the internal carotid artery to be occluded; the aneurysm was smaller. Another patient with an aneurysm of the middle cerebral artery, who had had ligation of the internal carotid artery, had a second hemorrhage and the arteriogram showed the clamp to be incompletely closed. These cases emphasize the importance of complete ligation.

The arteriogram does not tell the whole story, especially in patients with large aneurysms, and the clinical signs and symptoms must be considered. In 1 patient having a large aneurysm of the internal carotid artery, the progress arteriogram showed no aneurysmal lumen. Shortly before the progress arteriogram, the patient had a recurrence of frontoparietal headaches which continued intermittently over a period of 5 months, when they became more severe and more frequent. One month later, the patient had a massive subarachnoid hemorrhage and died within an hour. The rupture was found at the neck of the aneurysm, where a small lumen was also found. Over nine-tenths of the aneurysm was filled by organized thrombus and old clot.

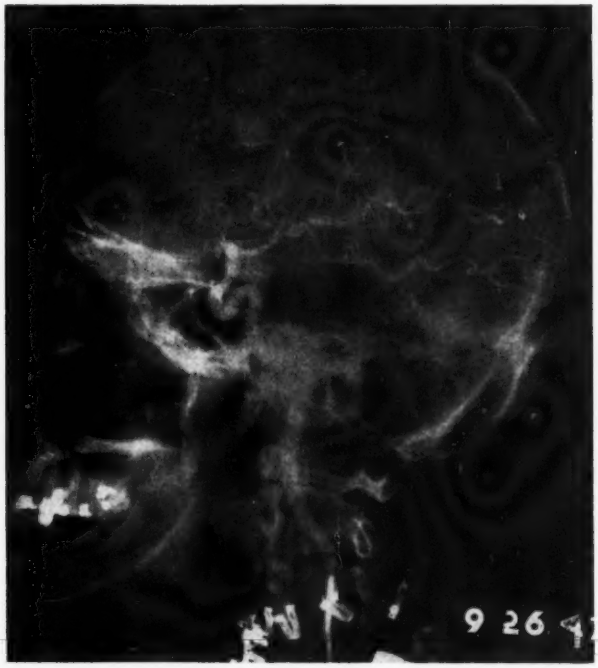

FIG. 4. Arteriogram showing a large aneurysm of internal carotid artery with upward displacement of middle cerebral artery.

FIG. 5. Progress arteriogram (same case as in Fig. 4) 7 years after ligation of internal carotid artery, which was shown to be incomplete, reveals no aneurysm of the internal carotid artery but the middle cerebral artery remains elevated.

Another patient, who had a large aneurysm of the internal carotid artery (Fig. 4), was treated by ligation of the internal carotid artery with a tantalum clip. Seven years later he began to have a recurrence of headaches. A progress arteriogram was done which revealed that the clip was partly open, but the aneurysm did not fill (Fig. 5). In spite of this, it was believed that there was some activity in the aneurysm, and the common and external carotid arteries were ligated with immediate relief of the headaches.

DISCUSSION

It is important to state that there were no complications as a result of the progress arteriogram, except in 1 patient who had a temporary paresis of one upper extremity; and further, that the arteriogram shows the status at the time it is done, and shows what has been accomplished. A negative arteriogram does not mean that the patient will have no trouble from the aneurysm in the future. The number of cases reported is not sufficient to draw any statistical conclusions. However, certain inferences are suggested and many interesting facts were found. The results of direct intracranial attack on the aneurysm, as demonstrated arteriographically, were found to be better than any form of ligation of the carotid arteries in the neck, in this small series. However, in 1 case the clips had loosened from the anterior cerebral artery and the aneurysm was the same size. In four-fifths of the patients with aneurysm of the internal carotid artery who were shown to

have complete ligation of the internal carotid artery, the aneurysm no longer filled on arteriography, and in the other one-fifth, the aneurysms were unchanged. The results were less good, as far as non-visualization of the aneurysm is concerned, when the aneurysm was on the middle cerebral artery, but in all the cases of complete ligation of the internal carotid artery the aneurysm was not seen or was smaller. The results were also less good when the clamp on the internal carotid artery was found to be open or when it had to be released. There were 2 patients in the entire group of surgically treated cases in whom the aneurysm was larger, and in both of these the clamp or clips were found to be open. These facts emphasize the importance of complete ligation but it would be valuable to know why the aneurysm did not fill in some cases of temporary or incomplete ligation. One obvious explanation is that it required only a few hours in these patients for the aneurysmal sac to fill with clot. In 1 patient who was operated on intracranially, 5 days after carotid ligation in the neck, the sac was found to be four-fifths filled with clot. It is significant that in the 16 patients who had no surgical therapy, the progress arteriogram showed the aneurysm to be the same size or larger. In not one of these cases did the aneurysm fail to show.

SUMMARY

1. The text describes the results of surgical therapy in 51 patients having intracranial aneurysms as shown by 54 progress arteriograms.

2. Progress arteriography is not a completely definitive method of evaluation of results; the clinical symptoms and signs are important.

3. The series is too small to draw conclusions on a statistical basis; but certain inferences can be made.

(a) Arteriographically the results of direct surgical attack on the aneurysm are better than those of any form of carotid ligation.

(b) Arteriographically the results of complete and permanent ligation of internal or common carotid are better than partial or temporary ligation and, of the two, ligation of internal carotid is superior.

(c) Ligation of the carotid arteries in the neck, using a tantalum clip, is likely not to be a complete and permanent ligation.

(d) In 16 patients who had demonstrated aneurysms and had no surgical therapy, the progress arteriograms showed the aneurysm in all cases; in 13 it was the same size, in 3 it was larger.

The authors wish to express their appreciation to the members of the neurosurgical staff, J. Lawrence Pool, Director, and the neurological staff, H. Houston Merritt, Director, who have kindly permitted the inclusion of their cases.

THE USE OF SODIUM DIACETRIZOATE* IN CEREBRAL ANGIOGRAPHY

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SINCE a method of visualization of the cerebral vasculature by contrast media was introduced by Moniz, investigators have searched for a substance that would afford good contrast with a minimum of complications. The sarcogenic and histopathologic effects[8] of thorium dioxide (Thorotrast) have restricted its use in angiography, although it is still used to a limited degree because it affords good contrast with practically no irritating effect on the injected vessels.

After the introduction of iodopyracet (Diodrast) in 1939,[5] it was used extensively in the performance of cerebral angiography. Complications have been reported extensively, both clinically and experimentally. Alterations of the blood-brain barrier caused by the toxic action of this substance have been attributed to the concentration and volume used. Recent writers have emphasized the need for limiting the amounts and rapidity of the contrast injection.[11,14,18] These principles also have been applied by those advocates of Triurol (Urokon)[9] as a contrast agent. Sutherland and associates[15] recommended the use of a 25 per cent solution of Diodrast, stating that complications resulting therefrom were definitely fewer than those that follow the commonly used strength of 35 per cent. With a solution of 25 per cent they found no difficulty in visualizing the finer branches of the carotid or basilar tree, admitting, however, that the contrast attained was less striking than that achieved with 35 per cent strength. We have used this weaker solution of Diodrast but have been disappointed in the degree of contrast obtained with it. Hence, we have ceased to use it, assuming a possibly greater risk of complication in favor of better diagnostic visualization.

Recently, we have used a new contrast medium, sodium diacetrizoate, or Hypaque Sodium (sodium 3,5-diacetamido-2,4,6-tri-iodobenzoate), in a solution of 50 per cent, with which we have achieved excellent contrast and distinctness of individual vessels with the use of injections of only 3 to 5

* Kindly supplied by the Department of Medical Research of the Winthrop Laboratories, Inc., Evanston, Illinois.

† The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

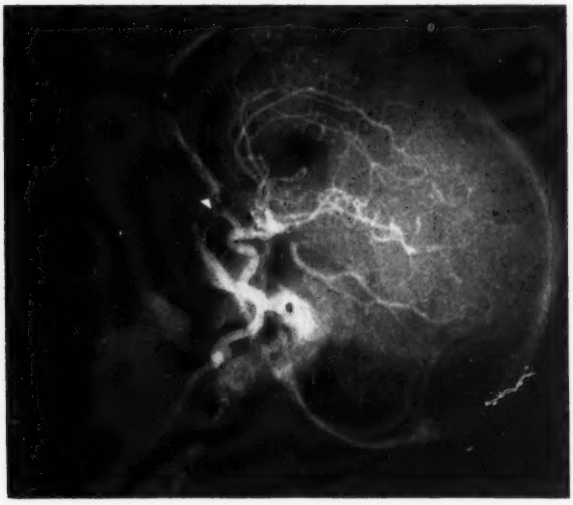

FIG. 1. Carotid angiogram in lateral projection made with 3 cc. of a 50 per cent solution of Hypaque Sodium.

cc. (Fig. 1). This synthetic compound contains 59.87 per cent iodine, with a pH range between 7.0 and 7.5. Thus far we have made 50 angiograms with the aid of this new substance, without serious complications. The ages of the patients ranged from 6 through 68 years. The diagnostic problems have included a variety of disorders, from brain tumor to occlusion of a major

FIG. 2. Massive petechial hemorrhages over the side of the face and bulbar conjunctivae after the injection of a 50 per cent solution of Hypaque Sodium.

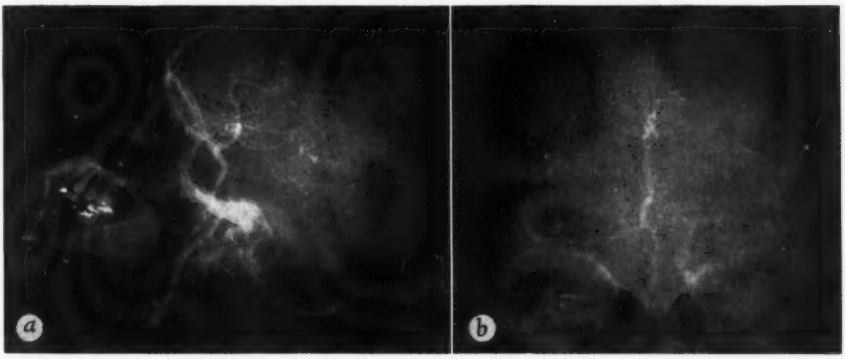

FIG. 3. (*a*) Angiogram made with a 35 per cent solution of Diodrast. The left internal carotid artery in the region of the sella turcica is distorted by a parasellar mass. The contrast obtained in this angiogram is considered to be typical of the experience of the authors with a 35 per cent solution of Diodrast. (*b*) Carotid angiogram, showing slight displacement of anterior cerebral artery to the right with elevation of carotid bifurcation. Excellent contrast was obtained with 3 cc. of a 50 per cent solution of Hypaque Sodium.

branch of the middle cerebral artery. Percutaneous vertebral angiography has been done in one instance, with good visualization of the basilar system. The only complication of the technic to date has been the development of extensive petechial hemorrhages over the face and orbital conjunctivae in a 36-year-old man with a definite history of allergic diathesis, but in whom no untoward reaction occurred during testing with 2 cc. of Hypaque Sodium administered intravenously 12 hours prior to angiography (Fig. 2). We have seen this degree of petechial formation very rarely as a complication of cerebral angiography carried out with a 35 per cent solution of Diodrast.[16]

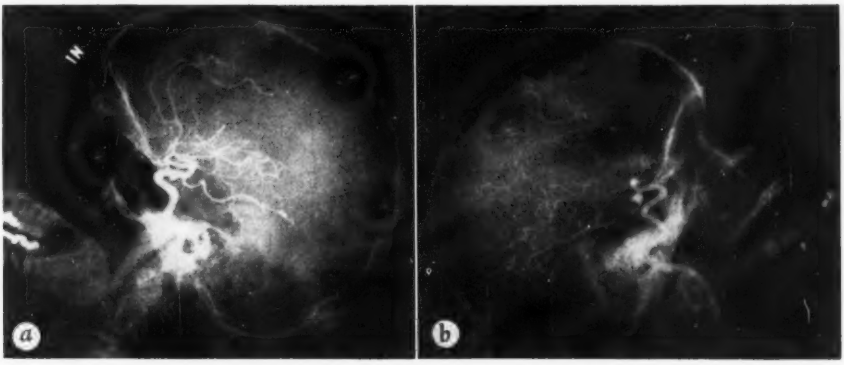

FIG. 4. (*a*) Excellent contrast in a film which demonstrates a small infraclinoid aneurysm on the right internal carotid artery. Filling of the right ophthalmic artery also is seen. A single injection of 3 cc. of a 50 per cent solution of Hypaque Sodium was used. (*b*) Excellent visualization of a berry aneurysm on the left internal carotid artery in the region of the posterior (superior) communicating artery. The contrast medium was 4 cc. of Hypaque Sodium.

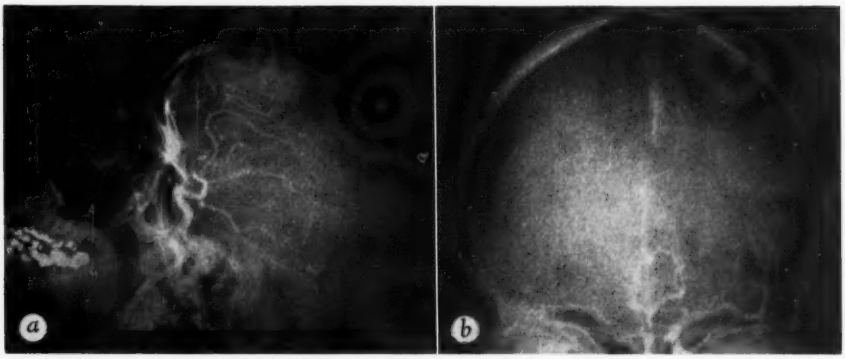

FIG. 5. (*a*) The left middle cerebral artery is elevated and curves over a mass in the left temporoparietal region. The contrast medium was 3 cc. of Hypaque Sodium. (*b*) In the half-axial anteroposterior view, there is little if any displacement of the anterior cerebral artery. Visualization obtained with 5 cc. of Hypaque Sodium.

The contrast obtained has been excellent. Filling of the smaller arterial radicals has been striking to even the most casual observer. The apparent increase in contrast density and distinctness of filling in individual vessels has resulted in more accurate diagnosis in the studies thus far performed (Figs. 3–6).

TECHNIC

The method of carotid angiography used at the Mayo Clinic has been described elsewhere.[16] The only modifications in technic have been necessitated by the smaller amount of contrast medium used in angiography with Hypaque Sodium. For lateral

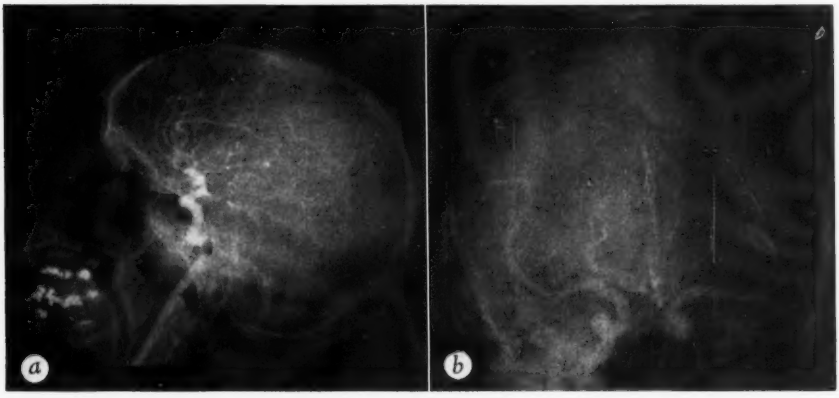

FIG. 6. (*a*) A small mass lesion in the right superior midtemporal region producing some stretching and elevation of the terminal branches of the middle cerebral artery on that side. The contrast medium was 4 cc. of Hypaque Sodium. (*b*) Anteroposterior view, showing excellent cross-filling in this case obtained with 5 cc. of Hypaque Sodium. There is a slight shift of the anterior cerebral artery across the midline to the left.

projections in the average patient, exposures are made at 150 mA., 100–110 PKV, with a tube distance of 30 inches and an exposure time of 1/30 second. With the Strifert angiographic machine, five lateral projections are made. The first three are taken immediately after completion of injection of the medium and are exposed at half-second intervals. The fourth and fifth exposures are done at 4- and 6-second intervals after the original exposure. In the half-axial anteroposterior projection roentgenographic factors are the same as for the lateral views, except that an exposure time of 1/20 second is used. Two exposures are made, the first being carried out after the injection of Hypaque Sodium has been completed, and the second, 1 second later.

COMMENT

Since there has been no change from older technics in our manner of performing angiography with Hypaque Sodium we feel that our assumption of a definite increase in contrast from the use of this new substance is valid. Smolik and Nash[13] have demonstrated the greater intensity of contrast obtained with a 50 per cent solution of Hypaque Sodium by an ingenious radiographic comparison of it with a 35 per cent solution of Diodrast and a 35 per cent solution of Urokon. Certainly, the observation of increased distinctness of individual vessels must, of necessity, be purely subjective.

Of interest is the fact that we have found that very good venograms can be obtained in the 4- or 6-second lateral exposure. The smaller amount of contrast medium used at the original injection of Hypaque Sodium does not seem to impair venous visualization. Whereas, we found it necessary to use at least 10 cc. of a 35 per cent solution of Diodrast to secure good visualization in the lateral projections, we have been able to secure remarkable contrast and more extensive visualization of the vascular bed by the use of only 3 to 4 cc. of Hypaque Sodium. In the anteroposterior projection we have obtained excellent contrast from the use of only 4 cc. of Hypaque Sodium, and for anteroposterior cross-filling we have used not more than 5 cc., as a rule. When we used a 35 per cent solution of Diodrast in these projections, we had to employ at least 12 to 14 cc. of the medium to approach the contrast obtained with Hypaque Sodium.

It is apparent, then, that complete arteriographic examination can be performed with as little as 16 to 20 cc. of Hypaque Sodium, as compared to 50 to 60 cc. of a 25 per cent or 35 per cent solution of Diodrast. Smolik and Nash[13] have used much larger amounts of Hypaque Sodium for single injections (12 cc.) and in total volume per study (average, 44 cc.). Undoubtedly, the contrast and filling under such circumstances would be even better, based on our results with much smaller volumes.

Another interesting observation is the fact that excellent visualization occurs even when both the external and internal carotid systems participate in the uptake of contrast medium. It has been our experience that when a 35 per cent solution of Diodrast is used, the degree of intracranial contrast is greatly reduced unless the puncture needle is deliberately placed in the internal carotid artery or can be so placed within the common carot-

id artery that it either enters the internal carotid artery or the stream of the opaque medium is directed into it. When Diodrast is used under these circumstances the density of contrast will be markedly lessened unless the volume is increased or the needle is repositioned. We have been much impressed with the degree of contrast and definitiveness of vessels obtained with only 3 to 4 cc. of Hypaque Sodium in this particular type of visualization.

We have had the opportunity to use this new contrast medium for only 5 patients with the aid of local anesthesia alone. All the rest were under the influence of general anesthesia with Pentothal Sodium. It is our impression that when Hypaque Sodium is used, the discomfort to the patient is less than it is when Diodrast is employed. When Hypaque Sodium was injected unilaterally into the internal carotid artery there has been virtually no discomfort behind the homolateral eye. We have seen only one instance of pain in the face after the use of this medium. In this case, after the x-ray films had been developed, it was seen that both the external and internal carotid arteries had filled. Although there is evidence that the injection of Hypaque Sodium causes discomfort, the lessened intensity of this discomfort may be in some way related to the decreased volume of the medium injected, as suggested by Odén[9] in his observations on the contrast medium, Triurol.

Moore and Mayer[7] reported on the use of Hypaque Sodium for excretory urography in 210 cases. They commented on the excellent contrast and diagnostic value of this medium. They encountered no major complications from its use. Minor complications of nausea, vomiting, excessive salivation, flushing, dizziness, urticaria and muscle twitching occurred in 13.3 per cent of their cases. They routinely injected intravenously 33 cc. of a 50 per cent solution of Hypaque Sodium for urographic studies. Smolik and Nash[13] observed no complications in the making of 39 cerebral angiograms in which the average dose per case was 44 cc. Schultz[10] and Watson[19] have reported advantages from the use of Hypaque Sodium in angiography.

According to Moore and Mayer,[7] Hypaque Sodium is closely related chemically to Urokon Sodium, although it would appear that the toxicity of the former is 25 to 40 per cent less than that of Urokon. Experimental observations on alteration of the blood-brain barrier when Hypaque Sodium was injected into the rabbit, as opposed to the situation when Diodrast and Urokon were used, indicate very little evidence of extravasation of trypan blue after the use of 50 per cent concentrations of Hypaque Sodium.[6] The most prominent effect observed experimentally was respiratory embarrassment, which ranged from transient apnea to respiratory arrest.[16] This effect also has been observed experimentally in the dog.[17] In concentrations of 50 per cent, iodopyracet (Diodrast) was found to produce marked extravasation of trypan blue with less alteration when concentrations of 25 and 35 per cent were used. This is in keeping with previous observations.[1–4,12] However, iodopyracet (Diodrast) was found to produce little disturbance in

respiration. Urokon (sodium acetrizoate) was found to produce severe extravasation of the indicator dye, especially in a concentration of 50 per cent.

In studies on the toxicity of these various compounds in local tissues, Hypaque Sodium was found to be least irritating of all.[6] As to the depressant effects of Hypaque Sodium on respiration in the experimental animal, investigators point out that the volume of contrast medium employed in the rabbit is roughly 375 times as great as that used for a single injection in man. They suggest that the increased volume may possibly account for the respiratory effects observed. We have not been aware of any respiratory complications evident clinically in the patients for whom this medium has been used thus far.

Unlike other contrast media, Hypaque Sodium is a highly water-soluble chemical compound. This physical quality may in itself be of some value in lessening the possibility of complication and affording a more even distribution of opaque substance within the vessels filled. Undoubtedly, its ability to obtain excellent contrast and definition in very small quantities will be of some advantage, and may compensate for its somewhat greater content of iodine per unit volume and its recognized hyperosmotic effect.[6]

SUMMARY

This is a preliminary report of our experience with a new contrast substance, Hypaque Sodium. This medium appears to be suitable for cerebral angiography, and we have routinely obtained excellent visualization of the intracranial vessels by the use of only 3 to 5 cc. of this substance in single injections. To date we have not encountered any serious complications other than rather extensive homolateral petechial hemorrhages involving the face, neck and conjunctivae on the side of the injection. These petechiae appeared within 4–6 hours in 2 patients but disappeared in approximately 3 days' time. In no case was there development of a hemiplegia or hemiparesis following angiography with Hypaque, though 1 patient did have some paraphasia following angiography. However, it could not be determined definitely whether this paraphasia was present to a mild degree before angiography or not. The paraphasia, however, cleared completely after the brain tumor had been removed.

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APPLICATION OF MONOMOLECULAR FILTER TUBES IN BRIDGING GAPS IN PERIPHERAL NERVES AND FOR PREVENTION OF NEUROMA FORMATION*

A PRELIMINARY REPORT

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REGENERATION of severed axons across an autogenous nerve graft rarely succeeds, when the diameter is larger than 2 mm. This limitation in caliber is determined largely by the ability of extracellular fluid to diffuse into the graft and act as a nutrient. Matson, Alexander and Weiss[2] obtained axon regeneration across a 6 to 14 mm. gap in the tibial division of the sciatic nerve in *Macacus rhesus* monkeys by interposing aligning filaments. Multiple threads of nylon or tantalum were suspended in clotted blood and encased in an autogenous vein graft. In spite of good regeneration, axons failed to reach normal diameters, primarily because of scarring at the point of exit of the filament from the proximal nerve.

Recently Algire *et al.*[1,3] have reported use of monomolecular cellulose filters in animals to investigate the response of the homograft. The *in vivo* properties of this material warranted the investigation of its potential in peripheral nerve regeneration. H. A. Millipore is relatively inert in tissues. It permits free diffusion of extracellular fluid, but restricts the transmigration of cells by virtue of its 0.4 μ size of pore. It has one disadvantage in that it is exceedingly brittle unless reinforced.

Millipore† tubes measuring 6 cm. long by 3 to 4 mm. in diameter were fabricated. Reinforced filter material was rolled into cylindrical forms and the edges were bonded with either Gelva or Glyptal. The tibial division of the sciatic nerve of the feline was isolated and severed. In one series of cats, the proximal and distal nerve ends were introduced into snugly fitting tubes of Millipore for a distance of 1 cm. A gap of 2.5 cm. in the nerve was maintained by suturing proximal and distal segments to the muscle bed. In a second group of animals, the nerve stumps were capped with closely fitted, blind socks of Millipore. Operative areas were excised for gross and histo-

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† This material was generously supplied by Mr. John Bush of the Millipore Filter Corporation, Watertown 72, Massachusetts.

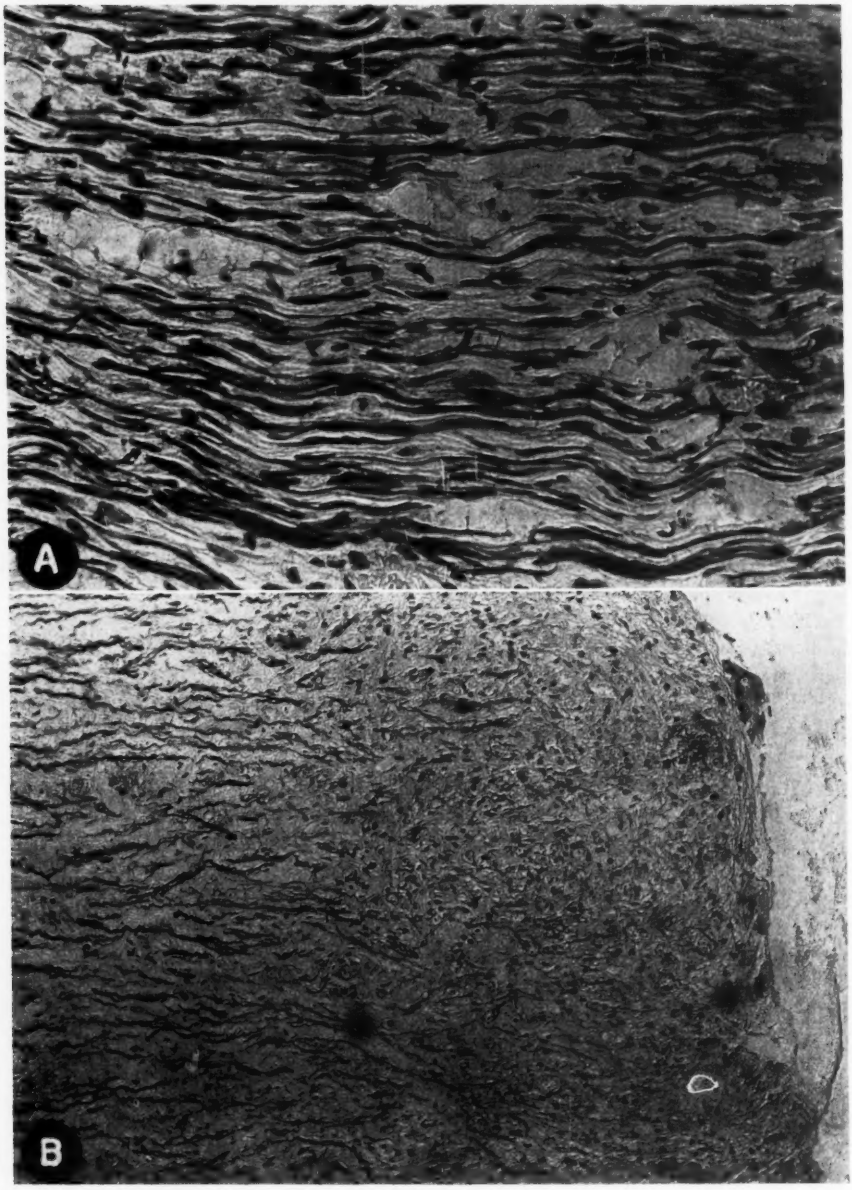

FIG. 1. (A) Distal segment of nerve, 10 weeks postoperative. Note recanalization of old Schwann tubules by regenerating axon. (B) Proximal nerve stump, 3 weeks postoperative. Note parallel alignment of axons, absence of scarring, and minimal tissue reaction to Millipore membrane. (Bodian stain)

logical examination 3 to 4 weeks postoperatively. However, in one animal with a gap, the Millipore tube was removed at 4 weeks and the nerve, with its bridging tissue, was returned to the muscle bed for an additional 6 weeks.

There was histological evidence of axon regeneration and envelopment by Schwann tubules at 23 days, and physiological evidence of regeneration at $2\frac{1}{2}$ months. Early myelin formation was apparent, and diameters of the axons in the proximal portion of the distal stump approached 1.8 μ (Fig. 1A). Rupture of the membrane before the gap was bridged resulted in classical neuroma formation by the intermingling of undifferentiated mesenchymal cells with regenerating neural tissue. If the filter was removed after bridging by differentiated neural elements had occurred, maturation continued without invasion of fibroblasts. Covering of proximal and distal stumps with blind Millipore socks prevented neuroma formation. Within the socks, orderly proliferation proceeded until the unobliterated space was filled by neural elements (Fig. 1B).

The method permits an initial burst of migration and proliferation of the neural elements in the proximal nerve stump. Cells are provided with a peripheral scaffolding of Millipore through which there is free diffusion of extracellular fluid for nutrition. Alignment filaments and clot have proven unnecessary so far, thus largely eliminating scarring of the Schwannoma type. The data justify preparation for clinical employment of the technic.

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CASE REPORTS

EXTRADURAL HEMATOMA OF THE SPINAL CANAL

REPORT OF A CASE

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Since Baine in 1897 first described extradural hemorrhage of the spinal canal, a total of 14 cases have been found in a review of the English literature. These cases have been well documented, individually, by Sadka.[2]

A positive etiology remains obscure, although in 10 of the 15 known cases, trauma seems to have played a role. The most logical explanation, and this is supported by the present case, is that an epidural venous abnormality or hemangioma is present which may rupture either spontaneously or traumatically.

FIG. 1. Myelogram showing the complete block at the 1st thoracic interspace.

It is felt that the diagnosis and treatment of these lesions may be difficult unless the progressive nature of the lesion is determined. It is obvious that the earlier surgery is instituted, the better the chance for recovery.

Guthkelch,[1] in reporting his 8 cases of extradural hemangiomas, had 1 patient with a remitting paraplegia. In the case presented below, there was also a remitting paraplegia.

REPORT OF CASE

A 15-year-old white male was referred by Dr. Wallace McNair of Aiken, S.C. on Feb. 1, 1956. His past history was significant only in that he had been having intermittent attacks of pain in the chest bilaterally for the past 7 months. This pain was not too bothersome, and he did not have to seek medical help. On Dec. 27, 1955, he was struck on the back of his head by the handle of a fertilizer spreader. This blow was sufficient to knock him down, but he did not have any sequelae from the accident itself. However, he noticed that the pains in his chest were more severe after the blow on the head. When the pain came it lasted approximately 5 hours and then would disappear. On the morning of admission to the hospital, he awoke with severe pain in his chest and marked weakness in his legs. He sought medical attention for this and was quickly referred to the University Hospital in Augusta, Georgia.

On arrival at the University Hospital, the patient stated that the weakness in his legs had passed away and that the pain in the chest was much improved.

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Examination. A mild symmetrical weakness in both legs was noted. The Babinski sign was positive bilaterally. There were no sensory changes and there were no posterior column signs. A complete blood count and urinalysis were normal. Temperature, pulse rate, and respiration were normal. Roentgenograms of the cervical and thoracic spine were normal. A lumbar puncture was performed which showed a spinal fluid pressure of 150 mm. of water. The fluid was clear and colorless. The Queckenstedt test was mildly suggestive of a block. The spinal fluid contained 3 white cells, a trace of Pandy, and a total protein of 28 mg. per cent.

Course. For the next 3 days, the patient showed gradual improvement, but continued to have a positive Babinski sign bilaterally.

At 4:00 P.M. on Feb. 3, 1956, severe pain in the chest again developed in the same distribution as he had been having for the past 7 months. This was accompanied by a rapid onset of paraplegia. There was a sensory level at T4. All modalities of spinal cord function were absent below this level.

Lumbar puncture revealed a pressure of 160 mm. of water. The Queckenstedt test showed a complete block. The spinal fluid contained 9 white cells, with a negative Pandy and a total protein of 40 mg. per cent. A myelogram revealed a complete block at the level of the 1st thoracic interspace (Fig. 1). The patient was taken immediately to the operating room.

Operation. A laminectomy was performed, starting at T1 and continuing up to C6 (Fig. 2). As the lamina was removed from T1, a dark bluish semisolid clot extruded under pressure. This hematoma was found to extend up to C6. While removing the hematoma, large tortuous veins were noted which bled freely. The mass was removed and hemostasis was secured. The dura mater was then opened and the spinal cord was visualized. The spinal cord was seen to be indented the length of the hematoma. This appeared to be of long standing. Complete

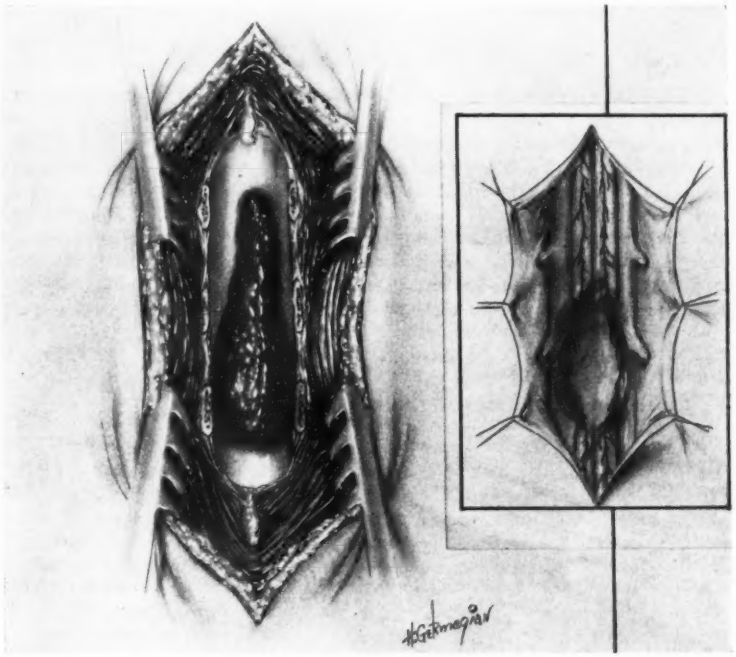

FIG. 2. Operative sketch showing the extradural hematoma and the compression of the cord beneath it.

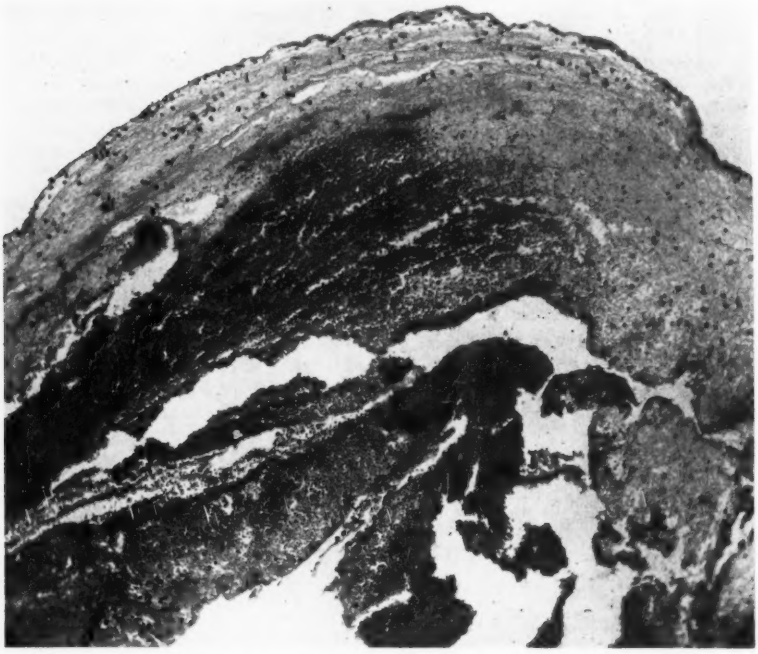

FIG. 3. Photomicrograph of hemorrhage enmeshed in fat (hematoxylin and eosin, ×120).

inspection of the spinal cord and spinal canal failed to reveal any other evidence of disease.

Pathologic Examination. The hematoma grossly consisted of blood enmeshed in extradural fat. The majority of the specimen was hematoma, but there was definitely a tangled mass of veins entering the clot. Microscopic preparation showed an organizing hematoma contained within fatty tissue, and a scattering of veins which were normal in appearance (Fig. 3).

Course. The patient made a rapid and uneventful postoperative recovery with complete return of spinal cord function. He was dismissed from the hospital ambulatory on the 9th postoperative day. Complete laboratory studies for any disturbance in bleeding and clotting mechanisms disclosed no abnormalities.

SUMMARY

It is felt that extradural hematoma should be considered in the differential diagnosis in any case of acute cord compression with absence of inflammatory signs or symptoms, particularly if there is a history of an injury.

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RECURRENCE OF CEREBELLAR HEMANGIOBLASTOMA AFTER 22 YEARS—OPERATION AND RECOVERY

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With the development of modern neurosurgery, the surgical treatment of cystic hemangioblastoma of the cerebellum can almost always be permanently curative. There are several reports of recurrent growth, however, with the longest recorded interval between the original operation and subsequent recurrence being 7 years.[3] According to Silver and Hennigar[4] such recurrences occur "because independent isolated foci manifested themselves at a later date, or because of continued growth of incompletely removed tumor tissue." The case presented here represents almost certainly a true recurrence of tumor 22 years after its original removal—a highly unusual situation.

REPORT OF CASE

A 42-year-old white man was first admitted to the New England Baptist Hospital on July 13, 1933, complaining of headache, vomiting and dizziness. These symptoms had been present for 10 weeks together with unsteadiness in walking. His past history was noncontributory.

Examination. Physical findings were normal, with normal urine and normal complete blood determinations.* Neurologically he showed fine nystagmoid movements to the right and left. The fundi showed early bilateral papilledema. There was some tendency to fall backward and to the right in the Romberg position.

On July 15, 1933, because of severe nausea and vomiting and extreme vertigo, the right ventricle was tapped. The fluid was found to be under high pressure and increased in amount, that is, there was a definite hydrocephalus. This drainage afforded considerable relief.

1st Operation. Three days later, on July 18, 1933, a bilateral cerebellar exploration was carried out by means of a "crossbow" incision. The operation was performed under procaine anesthesia, with the patient in the prone position. There was marked herniation of the cerebellar tonsils through the foramen magnum, and upon opening the dura mater over both cerebellar hemispheres it was apparent that the right hemisphere was larger than the left and that its convolutions were wider. The right hemisphere was likewise fluctuant to palpation. An incision was made horizontally through the widened convolutions, and 35 to 40 cc. of pale yellow fluid were evacuated from the underlying cyst. A bright red nodule of tumor was disclosed on the anterior superior wall of the cyst, lying laterally under the bony margin (Fig. 1). This nodule was excised completely, so far as could be determined grossly.

Course. Convalescence was normal for 6 days but then the temperature rose suddenly to 102.4°F. The median incision was reopened and an abundance of slightly yellowish fluid was evacuated at the cisterna magna. The wound was left open with gutta-percha tissue down to the bottom. Culture of the fluid showed hemolytic streptococcus. Convalescence was somewhat prolonged, but in the end was quite satisfactory and he was discharged on Aug. 20, 1933, with his wound well healed (Fig. 2). At that time he was up and about the ward, walking fairly well.

Pathological Report. The tumor was composed of capillary blood vessels in a matrix of connective tissue. Throughout the matrix there were varying numbers of endothelial-like

* This is important, in view of subsequent blood examinations.

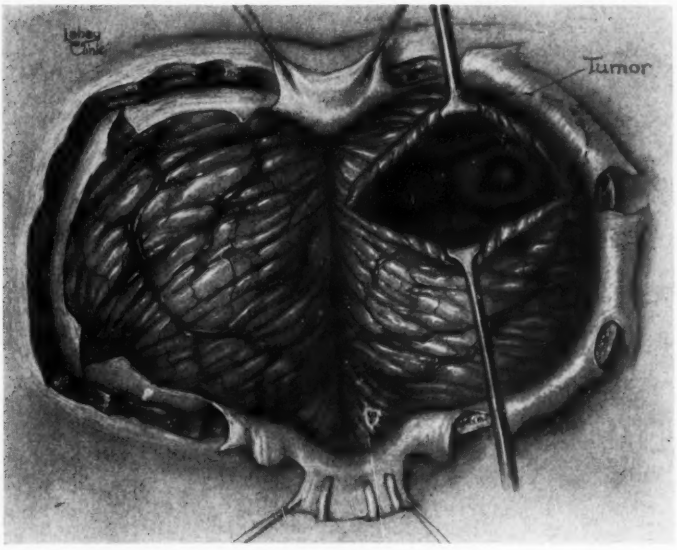

FIG. 1. Operative sketch of appearance of cyst and mural nodule at the time of the first operation. (Compare with Fig. 5.)

cells, some of which were rounded and resembled macrophages. These are sometimes called pseudoxanthomatous cells. In some areas cystic spaces had formed and occasional large vessels were seen (Figs. 3 and 4).

Interval History. After returning home, the patient made excellent progress and resumed his usual work on Feb. 5, 1934, 5 months after discharge. He continued in this way until he was normally retired at the age of 60.

In June 1955 he began to have increasing unsteadiness in walking, headache and soreness in the left suboccipital region. Because of the progression of the symptoms, the patient was admitted to the New England Baptist Hospital on July 6, 1955. A few months before admission a diagnosis of polycythemia vera had been made. This condition was treated by Dr. John W. Norcross of our medical department. On hematologic study the hemoglobin was found to be 25.5 gm., the red blood cells numbered 8,850,000, and the white blood cells 9,950, with a differential of 79 polymorphonuclear leukocytes, 14 lymphocytes, 1.5 eosinophils, 1.5 basophils, 0.5 young lymphocytes, and 1 atypical lymphocyte. The hematocrit was 70 and the reticulocyte count was 3.9 per cent with a platelet count of little over 1 million. The heart was enlarged roentgenologically with a cardiothoracic ratio of 16.4 to 32.3 cm. Because of the danger of a surgical procedure with the blood count as high as this, venesection was done on five occasions and his blood was brought to normal figures of 14.8 gm. of hemoglobin and 51 hematocrit before surgical therapy.

FIG. 2. The patient's wound at the time of his discharge from the hospital after his first operation. Note secondary closure of the median "crossbow" incision after it had been reopened for drainage.

2nd Operation. On July 15, 1955, the curved por-

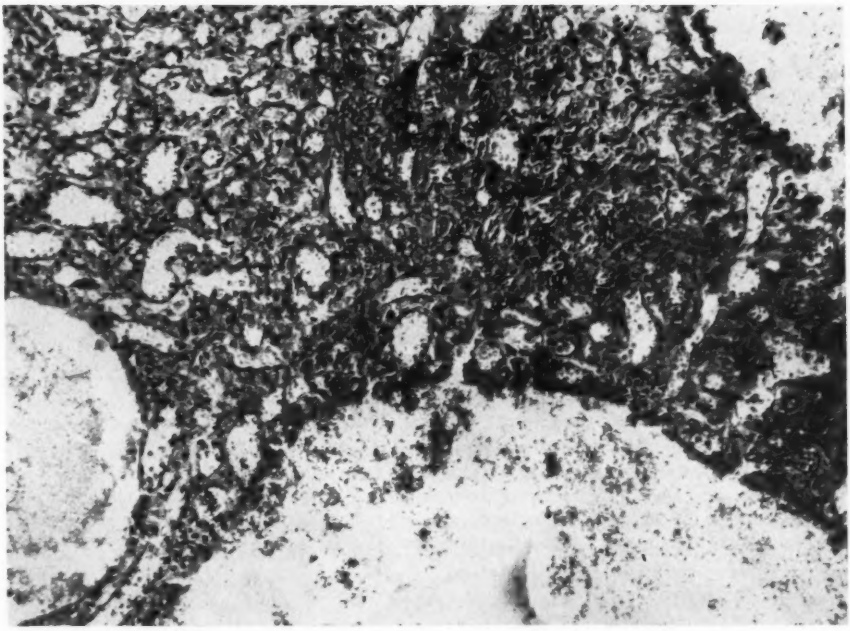

FIG. 3. Low power magnification (×130) of the tumor removed at first operation, to show general architecture with capillaries and cystic areas.

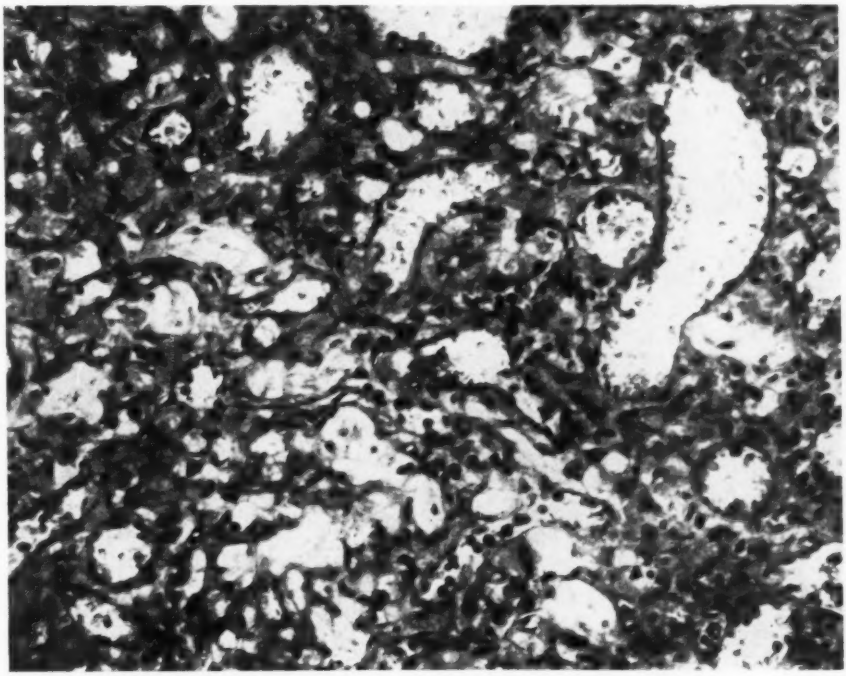

FIG. 4. High power magnification (×300) of original tumor. Note capillary blood vessels and typical xanthomatous cells.

tion of the old "crossbow" incision was reopened, with the patient in the sitting position under endotracheal ether anesthesia. Upon exposing the cerebellum, the left hemisphere appeared normal, but on the right side a considerable portion of the hemisphere was replaced by a large cyst, from which 30 cc. of canary-colored fluid were aspirated. Upon inspection of the smooth-walled cavity, a rather firm red nodule of tumor was seen in the exact situation from which the original mural tumor had been excised on the anterior, superior and lateral wall (Fig. 5). This tumor nodule was about 2×2 cm. in two diameters and was completely excised by means of electrocoagulation combined with suction. It impinged on the tentorium and there were large venous attachments from the lateral sinus. There were no particular difficulties, as might have been expected from the polycythemia.

Course. He made an uneventful recovery and was discharged on July 22, 1955, 7 days after the operation. At this time his red blood cell count was 4,710,000, his white blood cell count was 7,400, his hemoglobin was 14.9 gm., and his platelets numbered 655,000.

Pathological Report. Microscopic preparations of the tumor removed at this second operation showed essentially the same general architecture and features as those of the original tumor.

Subsequent Course. The patient was seen for a check-up examination on Aug. 4, 1955, and was making a normal convalescence. The polycythemia was checked intermittently after discharge and because his platelet count remained well above normal at about 650,000, on Sept. 16, 1955 he was given 5 millicuries of irradiated phosphorus by mouth in an attempt to reduce the count. His hemoglobin at that time was 15.4 gm., red blood cell count 5,800,000,

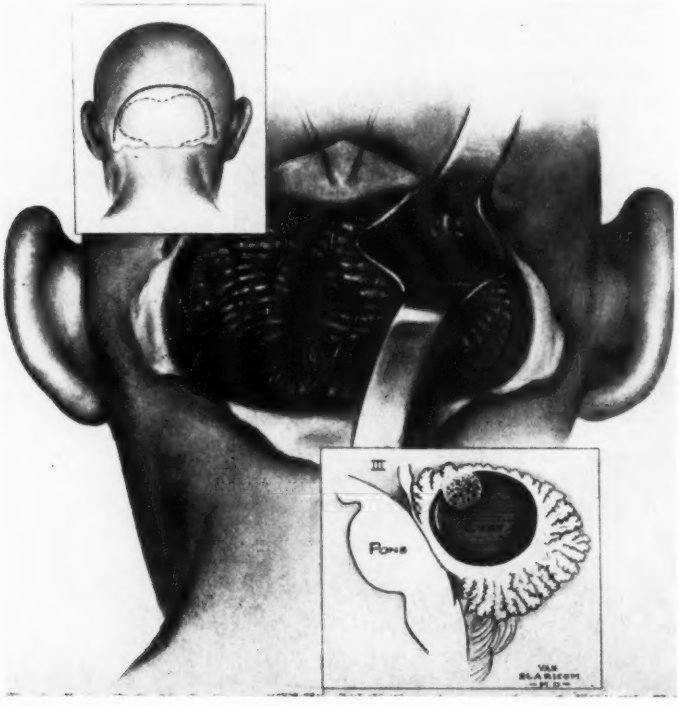

FIG. 5. Operative sketch of recurrent cyst and mural nodule in same situation as originally.

and hematocrit 47 per cent. His white blood cell count was 7,400 with a normal differential. The platelet count was reduced to 470,000. Subsequent blood studies on Dec. 16, 1955, were satisfactory.

COMMENT

Our chief interest in reporting this case is the long interval that elapsed between the time of the original operation and the recurrence of the cystic tumor, namely 22 years. That this second tumor represented a true recurrence is hardly open to question since the mural nodule was in exactly the same area as the original tumor. Furthermore, its microscopic appearance was similar to that of the original tumor. It must therefore be presumed that some tumor cells had been left at the time of the first operation.

A second point of interest lies in the associated polycythemia which had developed since this patient's first operation. The incidence of polycythemia in patients with recurrence was reported as 36.4 per cent by Carpenter *et al.*[1] Cramer and Kimsey,[2] however, found polycythemia in only 18 per cent of cases of recurrent tumor that they studied. They believe that this association may result directly from "hematopoietic functions of the hemangioblastomas or may represent a diasthesis toward extramedullary erythropoiesis in the spleen and liver." On the other hand, Carpenter and associates[1] stated that there is a neurogenic cause, the tumor stimulating "hematopoietic mechanisms in the diencephalon."

In addition to the features just mentioned, a brief word might be in order regarding the changing methods of neurosurgical procedures since the original operation on this patient in 1933. At that time the prone position and procaine anesthesia were utilized, whereas in 1955, the patient was operated upon in the sitting position and the anesthetic agent was ether given by the intratracheal method. Furthermore, in 1933 the Cushing "crossbow" incisions were used, but at the second procedure only the curved portion of these incisions was made use of since it has been recognized for many years that the median incision is unnecessary for adequate exposure, and much time and effort are saved by making only the "horseshoe" type of approach to the cerebellar region.

Although electrosurgical methods had been in use some 5 or 6 years before 1933, and this apparatus was used during the patient's original operation, nevertheless, techniques of electrosurgery have developed since that time, particularly the combined metal suction tube to which the electric current is attached. This instrument undoubtedly permits more rapid excision of a tumor such as the one in question, and perhaps by combining suction with coagulation there is less doubt as to the completeness of tumor removal. It may be noted also that in 1933 a piece of muscle was used to control venous bleeding from one of the vessels between the tumor and lateral sinus, whereas now either gelfoam or electrocoagulation would serve this purpose.

SUMMARY

In this case report, attention is called to the fact that a cystic hemangioblastoma of the cerebellum recurred 22 years after the original operation, at which time the mural nodule of solid tumor was apparently completely excised. At the time of the second operation, severe polycythemia vera had developed, a condition that has been noted in about one-fifth to one-third of the patients having recurrent tumors of this pathological type.

Certain interesting features concerning the developments in neurosurgery during the past 22 years are mentioned briefly.

The authors are most grateful to Dr. Samuel P. Hicks for his interpretation of the pathological material which was processed by the Department of Pathology of the New England Deaconess Hospital.

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TRIGEMINAL NEURALGIA AND ARTERIOVENOUS ANEURYSM OF THE CEREBELLOPONTINE ANGLE

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Trigeminal neuralgia has been reported as occurring very rarely in children. Grant,[2] however, has successfully treated by resection a 7-year-old boy, and our patient, now 32 years old, has a history of having typical trigeminal neuralgia that dates back to her twelfth year.

REPORT OF CASE

F.S.W., a 30-year-old woman, was admitted to the neurosurgical service of the Lakewood City Hospital in May 1953, with a history of right trigeminal neuralgia that had developed at the age of 12 and continued until 1951, at which time the symptomatology changed to a burning, searing discomfort in the same area, which remained as long as 5 minutes per paroxysm. That year, through a subtemporal craniectomy, rhizotomy of the right 5th sensory root was performed elsewhere. She continued to have ghost pains involving the entire side of the face, and headaches, which had always been present intermittently, became more frequent and severe.

Operation. Because of persistent facial discomfort, development of deafness in the right ear, ataxia and atonia of the right arm and leg, and nystagmus, a right suboccipital craniectomy was performed by one of us (W.M.H.), revealing a collection of large, tortuous, aberrant arterial vessels. These extended beneath the cerebellar hemisphere on the right side adjacent to the medulla and into the cerebellopontine angle. No attempt was made to resect the lesion because of its nature and location.

Course. The patient's symptomatology was not appreciably altered by operation; she was discharged from the hospital essentially unchanged and was lost to follow-up.

She was subsequently admitted to Cleveland Clinic Hospital on Jan. 21, 1955, 10 days after the acute onset of a sudden, loud noise in the head, "jumping and pulling" of the muscles of the neck, staggering gait, vomiting, headache, and stiff neck.

Examination. The patient was extremely apprehensive, and lumbar puncture at the time revealed xanthochromic cerebrospinal fluid. The blood pressure was 110 systolic and 70 diastolic; pulse rate and temperature were normal. The pupils were constricted and reacted only slightly to light. There was a fine nystagmus on lateral and vertical gaze. Funduscopic examination revealed early bilateral papilledema. There was no evidence of a vascular malformation in the retinal structures. Hearing was reduced in the right ear, and the Weber test indicated nerve deafness. Analgesia in the distribution of the right 5th nerve was complete, with paralysis of the muscles of mastication. The remaining cranial nerves were normal. The deep tendon reflexes of the right arm were notably diminished, but the patellar and Achilles reflexes were active and equal bilaterally. Unsteadiness in the Romberg position was pronounced and the gait was wide-based. The finger-to-nose and heel-to-knee tests were poorly performed on the right side. The Babinski sign was negative.

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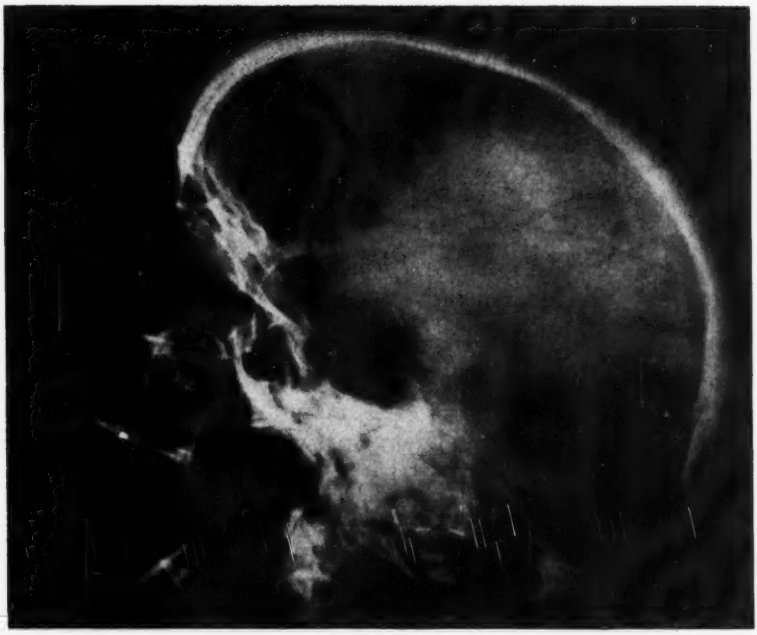

FIG. 1. Right lateral vertebral arteriogram of arteriovenous malformation producing trigeminal neuralgia.

Significant laboratory findings were xanthochromic cerebrospinal fluid at 220 mm. of cerebrospinal fluid pressure, containing 47 mg. per 100 ml. of protein and 3 cells. A right vertebral arteriogram revealed a large arteriovenous malformation in the right posterior fossa extending from the site of the suboccipital craniectomy to the level of the incisura and slightly above the tentorium cerebelli (Fig. 1). Carotid arteriograms filled only the upper extension of the malformation to a slight degree.

Course. No further treatment was undertaken. She remained in the hospital approximately 1 month, during which her neurological deficit was essentially unchanged. She became completely antagonistic and negativistic before discharge; she refused to eat and finally left against advice.

COMMENT

The occurrence of trigeminal neuralgia with lesions of the posterior fossa such as: cholesteatomas of the cerebellopontine angle,[6,8] acoustic neurinoma, meningioma and thromboses of pontine vessels and the posterior inferior cerebellar artery,[4] is well known. Olivecrona[6] also mentioned a case of arteriovenous malformation in which typical trigeminal neuralgia was the principal symptom. Dandy[1] stated that he had found 18 tumors in 250 cases of trigeminal neuralgia explored by the cerebellar route. In addition, he believed that free arterial loops elevating the nerve root, or venous branches crossing the nerve and sometimes dividing it, could account for the pain.

Peet and Schneider[7] found only 4 tumors in 689 cases of trigeminal neuralgia approached by the subtemporal route. One of those tumors was a true aneurysm.

Hamby[3] discussed aneurysm of branches of the internal carotid system as a cause of tic, and reviewed also the experience of several other authors.

Wyburn-Mason,[9] in a discussion of arteriovenous malformations of the brain, referred to the severe psychoneurotic and psychotic changes that sometimes are produced by these lesions. The question of whether the malformation was conclusively the basis of the pronounced personality changes demonstrated by our patient remains unanswered. She had admitted to having tried to "do away" with her children 2 years previously. Letters from her referring physicians emphasized the existence of a behavior problem.

Pneumoencephalography was not performed. However, on the lateral carotid arteriogram, stretching of the pericallosal vessels was present. The arteriographic findings, papilledema and increased intracranial pressure are indicative of an associated hydrocephalus.[5,9]

SUMMARY

An unusual case is presented in which there occurred trigeminal neuralgia with onset at age 12 years, subsequently found to be associated with and probably resulting from a large arteriovenous malformation of the right cerebellopontine angle, with hydrocephalus, a severely disordered personality, and cerebellar signs.

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PARTIAL INTERNAL CAROTID ARTERY OCCLUSION TREATED BY PRIMARY RESECTION AND VEIN GRAFT

REPORT OF A CASE

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The diagnoses of occlusions of the internal or common carotid artery have been made more frequently in the past decade.[3–6,8–10] The clinical manifestations are varied, and are dependent on the degree of obstruction. In most cases of thrombosis of the internal carotid artery, the lesion is associated with diffuse cerebral arteriosclerotic disease and occurs in the older age group. However, many cases have been reported in the younger age group.[3–5] The occlusion is usually caused by thrombus formation secondary to a localized arteriosclerotic plaque, frequently situated near the region of the bifurcation of the common carotid artery.

Most clinicians are aware of the presence of a pre-occlusive lesion of the carotid artery. However, other than a high degree of suspicion, there is not enough clinical material in the literature to describe it as a clinical entity. The following is a case of incomplete occlusion of the internal carotid artery treated surgically by resection and vein graft.

CASE REPORT

A 44-year-old sergeant was admitted to Madigan Army Hospital in December 1955 with the history of intense paroxysmal left upper facial pain since 1948. A typical headache was described as starting from the left upper temporal area radiating down to the preauricular region. This was followed by a piercing pain behind the left eyeball, and a shooting pain toward the left maxillary region and left nostril. The headache usually occurred during the night, with a sudden onset, and lasted from a few minutes to 2–3 hours. The patient stated that many times he was literally thrown out of the bed, when the headache suddenly seized him during sleep. Less frequently the headache would occur during the daytime. In a few instances, the headache was brought on after drinking cold water and liquors, though there was no definite trigger mechanism with most attacks. Intense emotional outburst would usually precipitate the headache occurring during the daytime. The headache was excruciating and was not relieved by opiates, ergot preparations or intravenous papaverine. The headache could not be precipitated by subcutaneous histamine injection.

During the past year, the attacks had become more frequent (1–2 a day), and more lasting. In addition to the facial pain described above, an aching pain on the left side of the neck and excessive lacrimation and scintillating scotoma of the left eye were noted. At the height of one of the typical attacks, one medical officer noted that the left pupil was dilated. For many hours after an attack, a definite Horner's syndrome could be found on the left. During the attack, and for 2 to 3 hours afterward, the patient also noticed a definite weakness on the right side of the body and difficulty in carrying out conversation. In the past 3 months, increasing weakness in the right arm was noticed.

Past history revealed that the patient had always been in excellent health. He appeared to be an intense and aggressive individual with a fine service record.

Examination. The patient was a white male of medium height, weighing 142 lbs. Blood pressure on admission was 130/80, pulse rate 78, and respiratory rate 22. The left eye showed slight ptosis and miosis (Horner's syndrome). Funduscopic examination showed no evidence of vasospasm or other abnormality. Visual field and extraocular movements were within normal limits, and corneal reflexes were active bilaterally. There was no sensory abnormality over the distribution of the left trigeminal nerve. Neurological examination of other cranial nerves revealed no abnormalities. The vessels of the scalp were not unusually prominent. Pulsation of the left superficial temporal artery was easily palpable and the artery was not tender. Definite weakness was found in the right hand grip. Some subjective weakness on the right side had been noticed by the patient, but other than the weak hand grip, the findings on examination of motor function were not remarkable. Deep tendon reflexes were hyperactive on the right side. The gait showed slight limping in the right leg. Babinski and Hoffmann signs were negative bilaterally. Sensations of pain, vibration and touch were normal. There was partial disturbance of the position sense and stereognostic sense on the right side. Handwriting, with eyes open and eyes closed, definitely indicated proprioceptive disturbance of the right hand. The proximal portion of the left common carotid artery showed weaker pulsation than its counterpart on the right. The left carotid pulsation could not be felt above the level of the thyroid cartilage. There was no enlargement of the cervical lymph nodes. No bruits were audible over the cervical region and left side of head.

Roentgenograms of the skull showed no abnormality. Serology was negative. Routine laboratory studies revealed no abnormalities. Spinal puncture showed a normal pressure of 160 mm. of water, and the spinal fluid was normal. Electroencephalogram showed slightly faster rhythms in all leads without any evidence of localized abnormality.

Left percutaneous carotid angiography (Fig. 1) was carried out with the needle inserted into the common carotid artery close to its origin. Three inches of common carotid artery

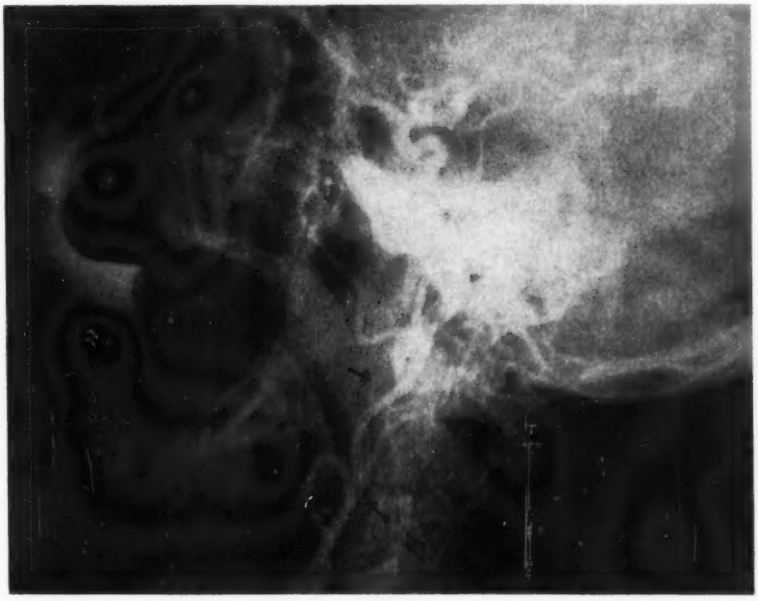

FIG. 1. Left lateral carotid angiogram. The arrows point to the marked narrowing of the proximal portion of the internal carotid artery.

FIG. 2. Appearance of the vein graft after completion of the anastomosis.

and all the branches of the external carotid artery were well visualized in all projections. However, in three successive injections, the proximal 2 inches of internal carotid artery could not be visualized even though the contrast medium was still present in the common carotid artery. The siphon and the middle cerebral artery were faintly visualized. In one lateral projection (Fig. 1) the marked narrowing of the proximal portion of the internal carotid artery was demonstrated. Right carotid angiogram, taken with contralateral compression of the common carotid artery, showed visualization of both anterior and middle cerebral arteries on both sides. There was a slight shift of the anterior cerebral arteries to the right. This finding was compatible with an atrophic lesion of the left cerebral hemisphere.

The clinical impression was that of a partial occlusion of the internal carotid artery, with increasing dysfunction of the left cerebral hemisphere and that the intermittent attacks of left facial pain could be explained on the same basis.

Operation. At exploration, a large arteriosclerotic plaque at the region of the carotid bifurcation was found. The arterial wall was greatly thickened near the carotid sinus. The thickening was so striking that it resembled a carotid body tumor. On palpation, partial occlusion of the lumen of the common carotid artery at the region of bifurcation was quite evident. The narrowing was more marked at the origin of the internal carotid artery, though the lumen at this level was not completely occluded. The internal carotid artery above this

level appeared to be of normal consistency. Resection of the common carotid artery including the bifurcation and the proximal 1 inch of the internal carotid artery was carried out. A 3-inch autogenous saphenous vein graft was used to bridge the defect between the common carotid artery and the internal carotid artery. The proximal end-to-end anastomosis between the common carotid artery and the vein graft was accomplished first. A continuous layer of 5-0 arterial silk was used for the approximation of the two ends. The external carotid artery was then ligated and severed. The distal anastomosis between the internal carotid artery and the distal end of the vein graft was then carried out. Before the completion of the distal anastomosis, e arterial clamp on the internal carotid artery was released to flush out clots or air bubbles. The total time lapse of complete occlusion of the internal carotid artery was 11 minutes, and that of the common carotid artery was 22 minutes. The wound of the neck was then closed in anatomical layers (Fig. 2).

Course. For 24 hours after surgery, the patient had slightly increased weakness on

FIG. 3. Percutaneous left carotid angiogram performed 3 months after the operation, showing excellent patency of the graft.

FIG. 4. Cross section of the proximal portion of the internal carotid artery. The lumen is narrowed by the arteriosclerotic process and compressed medially by the fibromatous thickened adventitia. An arteriosclerotic plaque of the intima can be seen medially. (×6)

the right side. This rapidly improved to the point where he thought he was stronger than before the procedure. Severe headache had completely disappeared. He complained of some sensation of pressure on the left side of the face and diminished sensations of taste and smell. A left Horner's syndrome became persistent after surgery. (The stellate ganglion and the left cervical sympathetic chain had been found to be unusually enlarged during the exploration, but were left intact.)

Left percutaneous carotid angiography done 3 months after the operation showed that the graft was patent. There was no evidence of aneurysmal dilatation or narrowing of the grafted segment (Fig. 3).

Pathological Report. Section showed arteriosclerotic thickening of the intima, especially near the carotid bifurcation and the proximal portion of the internal carotid artery. There was evidence of marked fibrosis with a few inflammatory cells embedded in the collagenous tissue of the adventitia and the muscular layers (Fig. 4).

Comment. The etiology of the fibrosis of the arterial wall is thought to be chronic inflammation or perhaps reactive changes associated with arteriosclerosis. The presence of abundant fibers of collagen in the tumor mass suggests chronicity. It is most unlikely that the percutaneous carotid angiography, which was performed only 10 days prior to the time of operation, could account for their formation.

DISCUSSION

Corrective attempts to rechannel an occluded internal carotid artery have been made by several workers.[1–3,7] Strully *et al.*[7] attempted thrombo-endarterectomy in a case of arteriosclerotic obliteration of the internal carotid artery, but were unable to completely rechannelize the internal carotid artery. Conley[1] has satisfactorily replaced portions of internal carotid artery and common carotid artery by vein graft in the treatment of tumors of the neck. Autogenous vein graft from the great saphenous vein has been shown to be a satisfactory substitute.

In the present case there were many of the earmarks of a typical migraine or unilateral histamine cephalalgia. The lability of the cervical sympathetic activity, pain on the left side of the neck, scintillating scotoma, transitory weakness on the right side during the attack, the marked diminution of carotid pulsation and the subsequent angiographic evidence of marked narrowing of the proximal portion of the internal carotid artery favored the preoperative diagnosis of partial occlusion of the internal carotid artery.

If relief of this patient's headache was the prime aim of therapy, one could indeed argue that perhaps a decortication of the common carotid artery or a stellate ganglionectomy would be sufficient. Though a definite focal partial occlusion at the bifurcation was established at the time of operation, the decision to resect a partially occluded internal carotid artery on the side of the dominant hemisphere was a bold one. Such a procedure might not have been undertaken, had it not been for the fact that despite adequate collateral circulation through the circle of Willis, there was still development of progressive deterioration of the left cerebral hemisphere with intractable left facial pain. Since it is well known that thrombosis occurs commonly in a markedly narrowed and diseased artery, a satisfactory result in a case of occlusion of the internal carotid artery could be accomplished only before irreversible cerebral changes had occurred.

It is not the purpose of this paper to advocate that the treatment of a partially occluded internal carotid artery should always be a primary resection. We merely wish to present a case of partial occlusion of the internal carotid artery with its in-

triguing symptomatology, and to demonstrate the feasibility of resection of a diseased segment of carotid artery and its satisfactory replacement by an autogenous vein graft.

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NOTICES

INTERNATIONAL CONGRESS OF NEUROSURGERY

The First International Congress of Neurosurgery will be held in Brussels, July 21–28, 1957, in conjunction with the International Congress of Neurological Sciences. Meeting jointly at that time will be the Sixth International Congress of Neurology, the Fourth International Congress of Electroencephalography, the Third International Congress of Neuropathology, the fifth meeting of the International League Against Epilepsy and the fifth meeting of the Symposium Neuroradiologicum.

During the first two days conjoint meetings will be held upon the themes of "Extrapyramidal Disease" and "Alterations in the State of Consciousness." On the subsequent four days, symposia will be held in conjunction with two or more of the Congresses on the subjects of "Multiple Sclerosis," "Supratentorial Angioma," "Hypothermia," "Petit Mal" and other topics.

A limited number of papers of neurosurgical interest may be accepted for presentation at the discretion of the Secretary-General of the First International Congress of Neurosurgery, Dr. Marcel David, Hospital Ste. Anne, Paris. Neurosurgeons wishing to submit titles for consideration may do so by sending a title and abstract of 200 words to the Assistant Secretary-General, Dr. William B. Scoville, 85 Jefferson Street, Hartford, Connecticut, who will forward them to the Secretary-General.

Registration forms, which must be completed before a paper may be considered for presentation, may be obtained from Dr. Scoville. Registration fees are as follows: Active Member, $15.00; Associate Member, $10.00; Member of the Women's Auxiliary, $5.00.

The American Express Company and Thomas Cook are the official travel agencies for hotel reservations and transportation facilities.

ISRAEL S. WECHSLER LECTURE

The annual Israel S. Wechsler Lecture is to be given on December 14, 1956 at 8:30 P.M. at The Mount Sinai Hospital of New York City, by Dr. Horace W. Magoun, Professor of Anatomy, School of Medicine, The University of California at Los Angeles, on "Aristotelian Psychology and The Twentieth Century Brain."

ITALIAN SOCIETY OF NEUROSURGERY

The 7th Congress of the Italian Society of Neurosurgery will be held in Milan on December 15 and 16, 1956. The subject of the meeting will be "Surgery of the Cranial Nerves." Papers will be presented by M. Milletti: Macroscopic and microscopic anatomy of tumors of cranial nerves; F. Migliavacca: Surgery of optic nerve; E. Miserocchi: Surgery of trigeminal nerve; G. Morello: Surgery of facial and intermediary nerves; P. Frugoni: Surgery of acoustic nerve; and F. Columella: Surgery of glossopharyngeal, vagus, spinal and hypoglossal nerves.

Secretary of the Society is G. Morello, Neurological Institute, Via Celoria, 11, Milan.

LATIN AMERICAN CONGRESS OF NEUROSURGERY

The 7th Latin American Congress of Neurosurgery will be held under the auspices of the University of Antioquia, Medellín, Colombia, July 8–11, 1957. The main subjects of the meeting will be (1) Ventricular Tumors: clinic, pathological anatomy, neuroradiology, treatment. (2) Temporal Lobe: physiopathology, epilepsy, tumors, radiology, surgery of tumors. (3) Apoplexy: clinic, pathological anatomy, radiology, surgical treatment.

Abstracts of papers should not exceed 400 words and must be sent to Dr. Ernesto Bustamante, Facultad de Medicina, Medellín, Colombia before April 1, 1957 so that they may be published in the program of the Congress.

NOTICE

Because of an unfortunate printer's error, the July 1956 issue of *Journal of Neurosurgery* (Vol. XIII, No. 4) contains wrong page numbers. The over-all pagination should be 311 through 412, instead of 223 through 324. The correct page numbers are:

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